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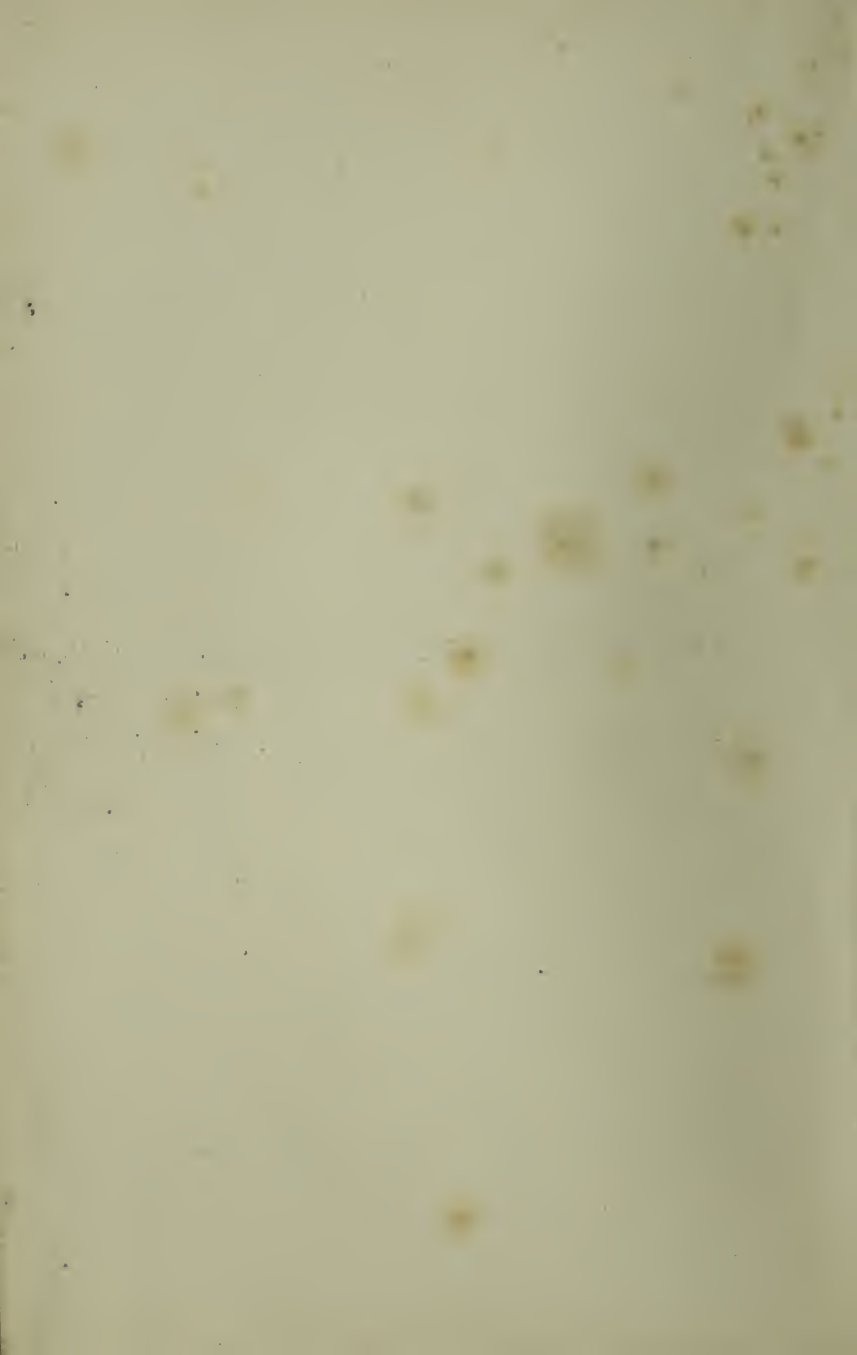
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ELEMENTS OF PRACTICAL MEDICINE



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OF
PRACTICAL MEDICINE

BY

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TO THE MEMORY
OF
MY FATHER
THE LATE
CHARLES HENRY CARTER, Esq., F.R.C.S.

THIS WORK
IS
AFFECTIONATELY INSCRIBED

“To know that we know that which we know, and to know that we do not know that which we know not, —that is true knowledge.”—*Persian Proverb.*

PREFACE TO THE TENTH EDITION.

THE ninth edition of this small work is now practically exhausted. From the fact that for thirty-two years it has been in fairly steady demand, I am encouraged to believe that, as an introduction to the study of systematic medicine, it has been found useful; and I have therefore undertaken the preparation of another edition, in order to bring it more nearly up-to-date.

It is surprising how soon a text-book of this kind falls behind current teaching; and no one is better able to appreciate this fact than an author who is called upon from time to time to critically review his work, at intervals of a few years. New facts require notice, traditional views need modification, and many opportunities are found of expressing old matter in newer—and possibly better—form. This has been largely so in the present case; and I have tried my best to make the book more worthy of the favour it has received, by careful revision of every page, and by re-writing and modifying much of the text.

Some additional matter has been almost unavoidable—not so much by a longer record of details, as by fuller treatment of the general principles on

which our knowledge of disease is based. Indeed, on reflection, I wish that I had dealt with the subject from the *general* standpoint to a still greater extent, with perhaps a further sacrifice of details. This conclusion is borne in upon me by considerable experience as an examiner in medicine. I have found that while the range of information displayed by candidates may give little cause for complaint, it is far too often so un-intelligent, so un-related, and so superficial, that it breaks down at once under cross-examination. If a student's reading during his earlier years of study were more restricted in range, there would be a better chance of infusing it with more intelligence and reflection ; and this is the opportunity of the elementary text-book.

In this edition, I have taken upon myself—perhaps unwisely—the whole of the re-editing, with the exception of the section on diseases of the skin. For this I am indebted to my friend Dr. Douglas Heath, in charge of the skin-department at the Birmingham General Hospital, to whom I tender my grateful thanks for his generous help. I must also again thank my publishers for the courteous consideration and kindly assistance which they have always shown me.

A. H. C.

BIRMINGHAM,
July, 1912.

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ELEMENTS OF PRACTICAL MEDICINE.

INTRODUCTION.

DISEASE may be defined as any disturbance of that balance between our bodies and their surroundings which constitutes normal health. It is essentially a dynamic process, which needs to be distinguished, on the one hand, from the causes which give rise to it, and, on the other hand, from any structural changes which are associated with it.

Disease is said to be *organic* when associated with obvious structural lesions to which it is causally related; while, in the absence of such lesions, it is said to be *functional*. The distinction, though convenient, is an arbitrary one; and many diseases formerly regarded as purely functional, are now known to be associated with definite organic lesions.

The term "disease" is also commonly employed in a more restricted and particular sense, as applied to certain groups of symptoms and physical signs, representing more or less definite types of morbid disturbance. The method of distinguishing individual diseases by conformity to a clinical type is both artificial and inadequate, and is adopted merely for convenience, until our knowledge has sufficiently advanced to enable us to define them in terms of causation. For instance, so long as pulmonary consumption was only known as a group of symptoms and signs it was difficult to distinguish it from other wasting disorders associated with lung changes; but on the dis-

covery of its dependence upon the growth and development of tubercle bacilli in the lung, it at once became possible to assign to it a precise nosological position.

Using the term "disease" in its restricted meaning, a distinction is drawn between *general diseases*, which affect the whole system (with or without focal lesions), and *local diseases*, in which the disturbance is primarily or mainly limited to some particular part or organ of the body. Diseases, again, are said to be *endemic* when they are peculiar to or are especially prevalent in certain districts; *epidemic*, when they spread rapidly among a large number of persons in one neighbourhood; *pandemic*, when they occur in epidemic form over whole countries or continents; and *sporadic*, when they occur as isolated or scattered cases.

CAUSATION.—Any abnormality either of the body itself or of its surroundings which interferes with satisfactory adjustment of the former to the latter may be a cause of disease.

The causes of disease may be either *inherited* or *acquired*. Some causes are of so violent and disturbing a nature that they set up disease-processes under any circumstances; yet more often this is not so. Either we find the chief or immediate cause associated with contributory causes in varying degrees of importance, or some condition of body constituting a special *predisposition* or *susceptibility* to the disease in question, in the absence of which it would not have occurred.

Disease can only be regarded as *inherited* when it is present as such in the fertile germ-plasm of the parent, before it becomes differentiated into the embryo. Any disease contracted later than this, up to the time of birth, is *acquired*, in the same sense as in after-life, though often distinguished as *congenital*. Heredity leads to disease only in one of two ways—either by imperfect or arrested

development, rendering the body incapable of proper adjustment to the conditions of post-natal life; or by impaired general or local vitality, creating a general or special predisposition to disease.

The causes of *acquired* disease vary widely both in their origin and nature. When they are related to surrounding conditions, or to substances introduced into the body from without, we speak of them as *exogenous*; and when of internal origin, they are called *endogenous*. The former are concerned with such factors as food, drink, light, extremes of temperature, infective or toxic agents, and parasites. Of these, infective agents are by far the most important, and will be dealt with in a later section. The endogenous group embraces such factors as anomalies of internal secretion; absorption of the products of disintegration of dead tissue-elements, or of decomposition of intestinal contents; and the results of perverted metabolism.

POWER OF RECOVERY.—The balance of bodily function which is characteristic of normal health is capable of resisting disturbance up to a certain point; and, when overthrown, tends to recovery within certain limits. Such native power is sometimes spoken of as the *vis medicatrix naturæ*, and varies much in different persons. But recovery may be favoured in many ways, among which may be mentioned the destruction of noxious agents within the body, expulsion and elimination of foreign substances, compensatory overgrowths, and gradual adaptation to new conditions. Treatment, broadly stated, consists of efforts directed either to the removal of disturbing conditions, or to the regulation, economy, and control of the bodily power which is possessed by the patient—efforts made in the hope that, under favourable conditions, the natural tendency to recovery will assert itself.

In such a view, there is nothing to encourage an attitude of non-interference; for the powers of resistance and

recovery, like other natural forces, are inexorable, with no purposive adaptation of means to ends, and are limited in their range of operation. Sometimes they are too feeble to be effective; at other times dangerous in their intensity. Or, again, they may be too slow to be of service; while accompaniments such as pain may interfere with their influence for good. Lastly, secondary disturbance may be set up, which, by unfavourable reaction upon the primary trouble, may lead to the establishment of vicious circles, which are often difficult to break through.

TREATMENT.—In order to bring the foregoing general principles into relation with actual practice, the first requisite is to acquire knowledge of the effects and modes of action of the various remedial measures at our disposal—not merely, or mainly, of drugs, but of those powerful agents known as “general remedies,” such as diet, heat, cold, rest, exercise, massage, baths, electricity, and climate.

Treatment should, whenever possible, be primarily directed to the *removal of the causes of disease*. At the same time, the *treatment of symptoms* has a very real and definite value. When we are ignorant of primary causes, or unable to remove them, it is obviously the only available method. Moreover, in a variety of ways, it may minister indirectly to recovery: namely, by the relief of pain and sleeplessness; by stimulation of depressed activity; by soothing over-excitement; by supplying materials needed by the body, such as iron, lime, and fats; by promoting elimination; and by attempts to obviate immediate tendencies to death.

Just a final word on the supreme importance of the personality of the practitioner in relation to the personality of the patient. Every endeavour should be made to obtain his confidence, to strengthen his will, to calm his fears, to encourage his hopes—in short, to enlist the co-operation of his whole moral nature in the work of his recovery.

SECTION I.

GENERAL PATHOLOGY.

CONGESTION.

By *hyperæmia* or *congestion* is meant a local over-fullness of the blood-vessels, which may arise either from a *too abundant supply* of blood to a part, or some *interference with its proper escape*. The former type of congestion is called *arterial*; the latter, *mechanical* or *venous*.

Arterial congestion.—Without entering into details which are beyond the present purpose, it is necessary to insist upon the close relation which exists between the behaviour of the tissue elements and that of the blood-vessels in this form of congestion, though the exact nature of the relation is as yet unknown. It seems clear from recent research, that, on the one hand, the tissue elements have the power of influencing the vessels in accordance with their physiological condition; and that, on the other hand, the vessels have the power of determining the nature of the response. When a tissue or organ is thrown into a state of functional activity under nervous influence, central or reflex, a demand is created for increased supply of nutriment, and also for the removal of the waste products of metabolism, to which the arterioles of the part respond—in some way unknown to us—by active dilatation, and a consequent increased supply of blood. The part becomes red, full, and (if superficial) is felt to be warmer. Moreover, the lymph-flow from the vessels to the tissue is increased

in proportion to the amount of activity which has been aroused, and local metabolism is more energetic. The vascular over-fullness which takes place under these conditions is what is termed *active congestion*. There is no difference in principle between the active congestion which thus characterises tissue in a state of ordinary functional activity excited by physiological stimuli, and that which occurs under morbid conditions from irritation of a mechanical, electrical, chemical, or thermal nature. So long as it is moderate in duration and intensity, no harm follows; but if severe, long continued, or frequently repeated, active congestion merges by imperceptible degrees into inflammation. In other words, active congestion is the result of a local tissue irritation which stops short of inflicting structural damage. Its distribution is always relatively restricted.

Another form of arterial congestion is the vascular over-fullness which is caused by atonic or paralytic relaxation of arterioles, such as results from the administration of certain drugs (*e.g.* nitro-glycerine), or from interruption of the paths of nervous control, in consequence of traumatic injury or disease. *Paralytic congestion* differs from the active variety in that it is commonly associated with reduction of the general blood-pressure, in consequence of which functional activity is subnormal, and the lymph-flow is *not* increased. Closely allied with this form is the arterial congestion occurring on the readmission of blood to a part previously rendered bloodless by prolonged compression.

A third variety is that which is known as *collateral congestion*, in which the arterial over-fullness is brought about by the diversion of blood from one part into another, caused by blocking or ligature of a large arterial trunk, or by obstruction of small afferent vessels over a considerable area.

Congestion arising from imperfect escape of blood is termed *mechanical* or *venous* when there is a direct impediment to the return of blood by the veins. It may be distributed over a wide area. It is a common result of uncompensated valvular disease of the heart, cirrhosis of the liver, direct pressure of a tumour upon important veins, and venous thrombosis. *Passive congestion* is closely allied to the foregoing, and is characterised by a sluggish and difficult circulation through the vessels of a part. It often occurs as a consequence of weakness of the heart, leading to accumulation of blood in dependent parts under the influence of gravitation, producing what is called *hypostatic congestion*.

Mechanical or passive congestion is characterised by a dusky or livid surface tint, with lowered temperature, diminished nutritive activity, and functional depression. Serious and persistent mechanical congestion is apt to lead to dropsy and hæmorrhage, and the affected part may ultimately become hard and dense from fibroid change.

HÆMORRHAGE.

Hæmorrhage denotes an escape of blood from the vessels, and may be occasioned by injury, disease, or faulty nutrition of the walls of the vessels, by increased pressure of blood within them, or by both of these causes acting together. Special terms are applied to hæmorrhage from particular parts: thus *epistaxis* signifies bleeding from the nose; *hæmatemesis*, from the stomach; *hæmoptysis*, from the lungs and bronchi; *melæna*, from the bowels; *hæmaturia*, from the urinary organs; *metrorrhagia*, from the uterus.

Hæmorrhage beneath a free surface forms more or less well-defined spots or small patches, which are called *petechiæ* or *ecchymoses*; and when blood is effused into a

tissue so as to form a tumour it is called a swelling, or *hæmatoma*. Those who are apt to bleed profusely from slight causes are said to exhibit the *hæmorrhagic diathesis*, or to suffer from *hæmophilia*.

Hæmorrhage occurs in two separate ways. There may either be a distinct rupture of the vascular wall, in which case the bleeding, whether large or small, takes place suddenly; or, as a result of partial detachment of the cells of the capillary endothelium at their edges, the solid and liquid elements of the blood may gradually ooze out (*diapedesis*), as seen, for instance, in hæmorrhagic infarction (page 19).

Blood which is extravasated and retained in the tissues, as a rule soon clots. The clot afterwards shrinks, the corpuscles disintegrate, and the liberated hæmoglobin undergoes chemical transformation, with changes of colour passing from brown to yellow. The residue may be altogether absorbed; or a reactive growth of new tissue may take place, ultimately forming a pigmented mark or cicatrix, with perhaps a few crystals of hæmatoidin; or, more rarely a cyst with fluid contents may result. Considerable hæmorrhage, attended with tissue damage, may be followed by pyrexia, due to absorption of the products of tissue-disintegration.

DROPSY.

Dropsy is a term applied to an accumulation of fluid lymph in the interstices of the tissues, or in the serous cavities of the body. Special terms are employed to indicate the extent and seat of dropsy. A generally distributed dropsy of the integuments is called *anasarca*; when localised, *œdema*; *hydrothorax* denotes dropsy of the pleura; *hydropericardium*, of the pericardium; and *ascites*, of the peritoneum.

Dropsical fluid has much the same general composition as that of blood-plasma, from which it is derived ; but its proteid content is much less, though it varies considerably in different dropsical effusions, according to their origin and seat. For the most part the fluid is of yellowish colour ; clear, or only slightly turbid ; alkaline ; and with a sp. gr. from about 1·008 to 1·016, according to the amount of albumin it contains. Inflammatory effusions may be usually distinguished by their turbidity from cellular elements in suspension, their tendency to spontaneous coagulation, and their higher sp. gr.—1·018 or over—due to the larger proteid content.

The pathological causes of dropsy fall into two main groups—namely, variations in the physico-chemical behaviour of the endothelial wall of the blood-capillaries ; and secondly, conditions leading to increased blood-pressure in the capillaries. It is very improbable that any increase of blood-pressure likely to be met with in disease is capable by itself of causing dropsy so long as the capillary wall remains healthy. On the other hand, bearing in mind the extreme delicacy of the capillary endothelium, its physico-chemical behaviour is almost bound to be modified by any change or impairment of its nutrition, with increased permeability as a likely result. If this be so, it is easy to understand that, while increased capillary blood-pressure is seldom or never a primary cause of dropsy, it may be a powerful factor in determining its amount and distribution.

Different pathological types of dropsy may be distinguished according to the circumstances under which they occur. (a) In the first type, *mechanical* or *passive congestion* is a leading feature. It is well illustrated by the dropsy which results from chronic cardiac or pulmonary disease, cirrhosis of the liver, or plugging of an important vein. Though the process is dominated by considerations

of blood-pressure, yet endothelial malnutrition from impure blood, languid circulation, and accumulation of waste products probably constitutes the first step. (b) The second type is characterised by the predominance of *toxæmia* or *general malnutrition*. The dropsy of chronic nephritis furnishes a good example of the former and anæmia of the latter. Quite frequently, however, these factors are supplemented by passive congestion due to a failing heart. (c) Another type is characterised by *inflammation*, where, as the result of damage directly inflicted upon the vessels of the affected area, an exudation of coagulable lymph takes place. (d) Lastly, cases are met with, in which the effusion is related in some obscure way to nervous disturbance (*neuropathic œdema*). It may be allied with changes indistinguishable from those of inflammation, such as we see in certain cases of neuritis. Sometimes, however, after certain foods, such as shell-fish, but often without discoverable cause, local areas of acute œdema rapidly develop on various parts of the body of a non-inflammatory nature (*angio-neurotic œdema*).

Dropsy may be either *general* or *local*; and *acute* or *chronic*. When associated with weak or impeded circulation, general dropsy usually begins in the most dependent parts; but when the main factor is a *toxæmia*, affecting the integrity of the vessel walls, it begins in those parts where the tissues are naturally relaxed, as the under eyelid, for instance. The integuments become swollen and pit on pressure. The vitality of dropsical parts is defective, and they are consequently liable to low forms of inflammation from slight causes. Should dropsy persist over a long period, especially when associated with venous engorgement, the tissues become brawny and indurated, and no longer pit on pressure.

DIAGNOSIS.—With regard to local dropsy it is necessary to bear in mind that when really local it must be due to

a local cause. General dropsy at its onset is usually limited to a particular part, but then it develops *symmetrically* on both sides of the body, and is steadily progressive.

The *acute form of general dropsy* may be distinguished by its abrupt onset, its frequent association with febrile symptoms, and its wide distribution. *Cardiac* or *pulmonary* general dropsy begins about the feet and ankles, gradually extends upwards, and is attended with the signs of disease of the heart or lungs. The skin is congested and dusky, and the superficial veins are more or less engorged. *Renal dropsy* is of two kinds. In tubular nephritis, for instance, it develops more or less quickly until it becomes general, beginning in the loose connective tissue of the eyelids or scrotum. The skin is pale and pasty, and the urine contains a large amount of albumen and tube-casts. In such cases interference with the nutrition of the tissues and capillary walls is the chief factor. In granular kidney, dropsy (when present) is usually of the cardiac type, beginning insidiously about the ankles or most dependent parts, relatively slight in amount, and develops slowly. The urine contains only a little albumen, and few casts. *Anæmic dropsy* is rarely extensive, and does not affect the serous cavities. It is usually confined to the ankles. Albumen is not often present in the urine, and the general anæmic condition is obvious.

TREATMENT.—This should be first directed to the removal of the cause. Absorption of the fluid may be aided by increasing the secretions of the skin, bowels, and kidneys, by diaphoretics, watery purgatives, and diuretics respectively. Finally, the dropsical fluid may be removed by surgical interference—by means of tapping (*paracentesis*) in the case of dropsy of the serous cavities ; or by punctures or incisions, in anasarca.

INFLAMMATION.

The process of inflammation represents those changes which take place in a living tissue as the result of local injury ; and therefore any agent capable of inflicting damage may become a cause of inflammation. It may result, therefore, from mechanical injury ; physical causes, such as extremes of temperature, corrosive agents, and the like ; or from the growth and development of micro-organisms. The last, perhaps, are the most common and important, and differ from other causes in the fact that, since they produce their effects by the poisonous substances (*toxins*) they liberate, their damaging influence is continuous and progressive, whereas other causes are of momentary or short duration.

The phenomena of inflammation vary according to the nature and intensity of the irritant, according to the resistant properties of the affected tissue, and according to the favourable nature (or otherwise) of the surrounding conditions ; but there is a broad similarity between them, and they may be conveniently considered under the following heads : (a) vascular disturbances, (b) exudation processes, and (c) changes in the inflamed tissues.

Vascular disturbances.—In the first place the arteries dilate and the blood-flow becomes accelerated (*active congestion*). This dilatation extends to the capillaries and veins of the part, and soon afterwards the blood-flow becomes retarded ; while the white blood-cells accumulate, and tend to adhere to the walls of the vessels. The current now becomes slower and slower, the vessels of the affected part become packed with corpuscles—both red and white—till at last complete stagnation occurs (*inflammatory stasis*). These disturbances are probably due to obscure morbid changes in the vascular walls.

Exudation processes.—Closely following the above events,

the white corpuscles—and the red also when the inflammation is severe—migrate from the vessels by diapedesis into the surrounding tissues, accompanied with plasma which exudes at the same time. The fluid and the corpuscular elements which have escaped from the vessels constitute the so-called *inflammatory exudation*. The nature of the exudation varies according to circumstances. When it speedily coagulates, it is said to be *fibrinous*; when, in addition, there is more or less serous effusion, it is termed *sero-fibrinous*, a common result of inflammation affecting serous membranes; while in inflammation of mucous membranes, the exudation is slimy from the presence of mucus.

Tissue changes.—In the first place there are changes characterised by *degeneration* and *necrosis*; and, secondly, there are changes characterised by *increased nutritive activity* and *proliferation*. It is impossible to analyse the exact relation of one to the other. Both represent the results of irritation, but while in the one case it is so slight as only to stimulate the tissue elements into a morbid activity, in the other it is so severe as to deprive them more or less rapidly of their vitality. At the centre of the inflamed area the degenerative and necrotic changes are most complete; while towards its periphery, only irritative changes are observed, representing the distant and weaker effect of the primary injury.

Course and terminations.—When inflammation is moderate, and due to a temporary cause, the vascular and tissue changes subside, and the exudation is absorbed (*resolution*). When more intense, the altered tissue elements and exudation degenerate, liquefy, and break down into *pus*, constituting what is known as *suppuration*.

Pus is an opaque yellowish fluid, of alkaline reaction, and average specific gravity of 1.030. It consists of an albuminous fluid (*liquor puris*), holding in suspension

innumerable small cells, closely resembling white blood-cells, in various stages of fatty change. Pus cells are mainly, if not entirely, derived from leucocytes which have migrated from the blood-vessels. A circumscribed collection of pus is called an *abscess*. When a limited surface undergoes molecular disintegration in association with suppurative change, an *ulcer* is formed. Very intense inflammation causes the death of tissue in mass; either forming a superficial *slough*, which separates after a time and leaves a deep ulcer behind, or leading to a deep-seated *gangrene*.

Repair of inflammatory mischief takes place in the peripheral zone of an inflammatory area, under the influence of stimulation sufficient to excite increased activity of tissue-nutrition, but inadequate for actual damage. The stimulus in question, though not forming an essential part of the inflammatory process, must be regarded as the most important of its secondary effects. Repair takes place for the most part by the proliferation of connective tissue cells, and a new formation of capillary vessels, which together form a delicate *granulation tissue*, undergoing subsequent organisation into fibroid tissue. Epithelial structures are also repaired by the formation of new cells, corresponding in type with those that have been destroyed, provided the deeper germinal layer retains its integrity. In the case then of connective tissue and epithelium, repair and regeneration are synonymous. On the other hand, highly specialised tissues such as muscle, nerve, and gland are not regenerated in kind, but their repair is effected by the formation of ordinary fibroid tissue. The repair of bone occupies a sort of middle place, in that, although ossification of newly formed cells occurs, no true bone is formed. Neither inflammatory exudations nor leucocytes take any active share in tissue-repair.

VARIETIES.—Inflammation may be *acute* or *chronic*, according to its intensity and rate of progress; *adhesive*, *suppurative*, *ulcerative*, or *gangrenous*, according to its effects; *circumscribed* or *diffuse*, according to its limits; *infective* or *simple*, according to its character.

The varieties of inflammation in mucous membranes may be conveniently described here. Three forms are generally recognised, representing different grades of intensity and depth to which the inflammation extends. The first and mildest form (*catarrhal inflammation*) is characterised by congestion and swelling of the membrane, desquamation of epithelium, and increased secretion. The latter at first consists of liquid exudation from the vessels, together with mucus, and is rendered more or less turbid from mixture with shed epithelium. If the process continues, an exudation of leucocytes takes place, which gives a semi-purulent character to the secretion (*purulent catarrh*): and if severe, there may be erosions of the surface (*catarrhal ulcers*). At any stage the inflammation may subside; or, on the other hand, it may become chronic. In the latter case, the membrane becomes thickened by fibrous hyperplasia, and the character of the surface becomes variously altered. In the second form (*croupous inflammation*) the process is more severe, the epithelium is partially destroyed, and the vessels are so damaged that a fibrinous exudation takes place on the surface, forming a “false membrane.” The latter is readily removable, and has no tendency to become organised. In the third form (*diphtheritic inflammation*) the process is so severe that the epithelium is destroyed without desquamation, and becoming saturated with fibrinous exudation, undergoes a sort of coagulation, and forms a close-textured “false membrane.” If moderately superficial it is scarcely distinguishable from the croupous form: but if, as not infrequently happens, the process is

so severe as to also destroy the deeper layers of mucous membrane, the whole is involved in the exudation, which afterwards separates by necrosis or sloughing, and leaves a well-marked ulcer. In course of time the latter undergoes cicatrisation, and may be covered with new epithelium.

Inflammation is manifested locally by redness, swelling, and heat; and is accompanied with pain, throbbing, tenderness, and disturbance of local function. There is also more or less pyrexia, in proportion to the degree and extent of inflammation; and, if suppuration occurs, the fever is usually of a hectic type.

THROMBOSIS.

Thrombosis denotes coagulation of the blood in the heart or vessels during life. The clot is called a *thrombus*. The factors on which thrombosis proximately depends are essentially the same as those which cause blood-coagulation elsewhere; but the structure and appearance of a thrombus varies considerably according to the circumstances under which it is produced. For instance, when it is recent, and formed when the blood is stationary—as after ligature of a vessel—there is nothing to distinguish it from the ordinary clotting of blood after death. On the other hand, when it is formed slowly from blood in motion, it is paler, firmer, often granular in texture, adherent to the parts with which it is in contact, and may present a laminated structure.

Among the circumstances which favour thrombosis, *retardation of the blood-current* may be mentioned first. It may result from cardiac weakness; or from ligature, compression, narrowing, or plugging of a vessel; or from aneurism. It will be found that thrombosis is more common in the veins than in the arteries. In complete stasis, coagulation occurs at once, as the result of enzymes

liberated by disintegration of blood-cells reacting upon the blood-plasma. Slowing, short of stasis, only acts as a predisposing factor, in co-operation with others. A further important factor is provided in many cases by *injury or disease of the vascular wall*. Here the fibrin-forming enzymes are yielded either by disintegration of damaged tissue-elements in the walls of the vessel, or by blood-platelets and leucocytes adhering to the altered surface. Lastly, thrombosis may be due to *the presence in the blood of microbic or endogenous toxins*. In one or other of these ways, acting alone or in combination, most—if not all—cases of thrombosis may be accounted for.

When the thrombus is small, it may be completely absorbed, and the circulation be re-established. More often it either becomes *organised* into fibrous tissue by the extension of vascular granulations from the wall of the vessel; or it undergoes *simple softening*, beginning at the centre and extending outwards. The latter is more common in cardiac thrombi, which in this way may be converted into little bags of thick creamy fluid. More rarely, the thrombus undergoes *calcification*, forming a concretion called a “phlebolith.” If associated with septic conditions, it undergoes *purulent* softening, forming an abscess, in which the coats of the vessel become involved.

When the thrombus becomes organised, the coats of the obstructed vessel become thickened and indurated, and the vessel is converted into a fibrous cord. Certain circulatory disturbances also occur:—thus, unless collateral circulation be quickly established, thrombosis of an artery will lead to the death of the parts supplied; while thrombosis of an important vein will cause swelling, and—if long continued—nutritive changes in the part which it drains. Fragments of the clot are liable to be washed off into the current of blood, and lead to embolism.

EMBOLISM.

Embolism denotes the plugging of a vessel by a substance carried to it from a distance. The plug is called an *embolus*. The most frequent sources of emboli are detached fragments of thrombi in the veins; vegetations and blood-clots from the heart; drops of oil which have escaped from fat-cells after certain surgical injuries, etc., and become absorbed by the veins; or portions of new growths; or parasites.

An embolus, arising in one of these ways, is swept into the general current of the circulation, in which it flows on until its further progress is arrested by a vessel the calibre of which is too small to allow it to pass. Thus, emboli which originate in the systemic veins, or on the right side of the heart, will be probably arrested in one of the smaller branches of the pulmonary artery; while those originating in the pulmonary veins, or on the left side of the heart, will be lodged in one of the systemic arteries; and those from the radicles of the portal vein—in the liver. Embolism may occur in any part of the body; but its commonest seat is in the spleen, kidney, brain, or lungs.

When an artery becomes plugged by embolism, the blood is obviously at once cut off from the area supplied by the vessel. The further results vary according to circumstances. When the embolism is small, or in larger embolisms when collateral circulation is readily and quickly established, the functions of the part are only temporarily disturbed, nutrition does not suffer, and complete recovery soon takes place. When, however, the obstructed artery is terminal—that is to say, with relatively few anastomoses—the functions of the part are suspended or abolished, the vessels of the area become engorged by overflow from surrounding capillaries, followed by a gradual escape of

blood-cells by diapedesis (*not* by rupture), eventually causing a hæmorrhagic infiltration of the area, which is in this state called a *red* or *hæmorrhagic* infarct. Such an infarct is usually conical in shape with a fairly defined margin, dark-red on section, and surrounded by a zone of congestion. It next undergoes coagulation-necrosis (in a degree inversely proportionate to the freedom of its blood-supply) and becomes steadily paler. On this account, an infarct in this pale stage has been called a *pale infarct*. [Recent advances in our knowledge of the subject would suggest the use of this term in a more specific sense. The progress of an infarct would seem to depend not merely on questions of blood-supply, but on the rapidity with which death of the tissue-elements takes place in the area involved. In a delicate tissue like the cortex of the kidney, for instance, where infarcts are specially pale, it is said that coagulation necrosis (page 29) occurs early enough to prevent the capillary engorgement which gives rise to hæmorrhage; and that thus the infarct is *white* from the beginning.] Meanwhile, the area becomes encapsulated by granulation tissue; the infarct shrinks from gradual absorption of the products of disintegration; and, ultimately, a depressed and puckered scar is all that remains to indicate what has occurred. Minor variations from the above account arise out of structural differences in the affected organs.

When the embolus is infective, or, in other words, if it be derived from an infective source, as from a thrombus occurring in the neighbourhood of an abscess, rapid softening and disintegration of the infarct may take place, resulting in the formation of an *embolic* or *metastatic abscess*, as in pyæmic.

HYPERTROPHY—HYPERPLASIA.

Hypertrophy denotes an increase in the mass of an organ or tissue, from overgrowth. When due to an absolute

increase in the *size* of the individual tissue element, hypertrophy is said to be *simple*, but when due to an increase in their *number*, it is termed *numerical hypertrophy* or *hyperplasia*. The distinction is not of much practical importance, as they are so often combined.

Pure hypertrophy implies *increased functional activity* on the part of the affected tissue-elements. It also necessarily assumes an accessible and adequate supply of nutritive material, without which it cannot take place. On the other hand, abundant nutritive supply cannot by itself determine overgrowth. Functional activity may be increased by a great variety of stimuli, always provided they are not carried so far as to exhaust or damage the part. In many cases the stimulus takes the form of physical strain, or increased work. Thickening of the inner coat of the arteries in certain forms of degeneration, attended with weakening of the middle and outer coats, furnishes an example of overgrowth from strain; and, in cardiac hypertrophy from valvular disease, or of the muscular wall of the bladder from chronic urethral stricture, we see the results of increased work. The increased muscular development of the athlete affords a purely physiological illustration of the same thing. In other cases increased functional activity is the result of nervous stimulation, as in the enlargement of the breasts during pregnancy; and, yet again, hypertrophy is of common occurrence in one of paired organs, like the kidneys, when the other has been removed or remains undeveloped; or in one of two organs functionally related, like the thyroid and pituitary bodies. In most of these cases it will be seen that hypertrophy is a protective or compensatory process, rather than a diseased condition.

Some forms of hypertrophy (or rather, hyperplasia), such as overgrowth of the lymph-glands in lymphadenoma, or of the bones in acromegaly, must be regarded as morbid in

nature, but we have at present no exact explanation of their occurrence.

Reference has already been made to the increased formation of fibrous tissue, in connection with inflammatory repair; and fibrosis (*fibroid hyperplasia*) is a common result of irritation, which falls short of inflammatory damage. It has been usual to describe this condition as one of "chronic" or "interstitial inflammation"; and examples are afforded by fibroid pneumonia, hepatic cirrhosis, and granular kidney. On the other hand, fibrosis occurs in the absence of inflammation or irritation, secondarily to antecedent degeneration of the elements of highly specialised tissues, such as nerve and muscle, or destruction of the secreting cells of glandular organs. The explanation of such fibrosis is obscure.

ATROPHY.

Atrophy implies a quantitative diminution of the substance of an organ or tissue, and is the outward sign of impaired functional activity. Atrophy may be regarded as *simple* or *numerical*, according as the tissue-elements are reduced in size or diminished in number; but the distinction is of no practical importance.

Of the various forms of physiological atrophy, senile decay is the most important. It is characterised by progressive exhaustion of natural vitality, associated with structural atrophy. The generative and lymphatic structures are ordinarily the first, and the central nervous system the last, to dwindle and decay. Fat cells shrink and lose their fatty contents, and the skin becomes wrinkled, partly from loss of subcutaneous fat, and partly from impairment of elasticity. Muscle-cells shrink and exhibit pigmentary deposits, giving rise to the condition known as *brown atrophy*. The bones undergo absorption

from within, and thus become lighter and more fragile. Loss of elasticity and atrophy of the lungs lead to emphysema; and, lastly, the arteries gradually lose their elasticity and contractility, may undergo calcification, and become converted into rigid, brittle tubes. Side by side with these atrophic changes in the specific tissue elements we find a tendency to progressive *fibrosis*. The advent of senility varies widely, and sometimes occurs prematurely as the result of inherited or acquired defect.

With regard to other forms of atrophy, we may recognise four varieties: namely, that which depends (1) upon defective nutrition, (2) upon disuse, (3) upon exhaustion from over-work, and (4) upon defective innervation.

General atrophy is a good example of the first group, and is due to *insufficient supply of nourishment*, as in starvation, constriction of some part of the alimentary canal, and defective digestion, assimilation, or absorption; or to *excessive waste*, as in fever, protracted diarrhoea, and immoderate hæmorrhage; or to some combination of these causes. In general atrophy the adipose tissue is first affected; then the muscles and viscera; and, lastly, the tissue of the nervous centres. Local atrophy, from diminished blood-supply or direct continuous pressure, comes under the same head.

The best example of the second group is the atrophy which takes place in muscles from prolonged disuse. The disappearance of the ductus arteriosus and the Wolffian bodies after birth, and the shrinking of the mammæ after lactation, furnish instances of physiological atrophy from disuse.

The third variety may be illustrated by the muscular atrophy which follows upon exhaustion of certain groups of muscles or their nervous centres, as the result of excessive writing, sewing, etc.

Atrophies from faulty innervation are classed as *tropho-*

neuroses, on the assumption that nutrition is largely controlled by nervous centres, from which trophic impulses are distributed along the nerves to the parts in connection with them. Any lesion of trophic nerve-cells, or their efferent prolongations, leads to rapid atrophy and degeneration of the tissue under their control. The muscular wasting in infantile paralysis, from lead-poisoning, and from division or injury of peripheral nerves, afford examples of this form of atrophy.

DEGENERATION.

Strictly speaking, degeneration differs from atrophy in that it is characterised by *qualitative* rather than by *quantitative* changes in the tissue-structures. At the same time, the two are so commonly associated that it is practically difficult to separate them.

Broadly stated, the various forms of degeneration represent types of perverted or retrograde metabolism, leading to the formation and accumulation in the tissues of materials less complex in composition, and of lower physiological rank than that which is proper to them.

Degeneration is always attended with impairment of function, though to a variable extent according to its kind. At one time it was usual to classify degenerations, as cell-degenerations proper, in which the cell-substance was primarily involved; and infiltrations, in which the newly formed material was merely deposited as such in the affected part, from without. It is now clear, however, that the various processes of degeneration are of far too complicated a nature, and far too little understood as yet, to admit of such simple classification. It will be better, therefore, simply to describe the principal types of

degeneration in turn, keeping as far as possible to the old nomenclature.

CLOUDY SWELLING is a form of degeneration to which the elements of various tissues and organs are prone, especially the muscles and the glandular epithelium of the liver and kidneys. It is a common feature of early inflammation, of pyrexial diseases, and some forms of metallic poisoning. Cloudy swelling is the outcome of metabolic changes taking place in the cell, as the direct effect of toxic impregnation. A good example of it is afforded by the condition of the renal epithelia shed in the early stages of tubal nephritis.

The affected cells are swollen and finely granular, but clear up on the addition of acetic acid. Muscular fibres swell up, and lose their striæ. The nucleus is hidden by the granular deposit, but is not destroyed. Cloudy swelling often merges into fatty degeneration.

FATTY DEGENERATION.—This form of degeneration is most frequently met with in the heart, liver, and lining membrane of the arteries. It is characterised by some increase in size of the affected part, pale colour, diminished firmness, and greasiness to touch. Microscopically, the fat occurs in the form of minute oil globules, fairly uniform in size, and with little tendency to fuse together. As the process extends, the cells or fibres increase in size, and may ultimately break up, in which case the albuminous matter between the globules liquefies, and the fat is distributed in the tissues. Fatty degeneration has long been regarded as representing a retrograde metabolism of the proteid cell-substance, with the conversion of its carbon-containing moiety into fat. Considerable doubt has of late been thrown on the correctness of this view, but it is too difficult a question to discuss here.

The formation of milk in the glandular epithelium of the breast, and of sebum in the cells of sebaceous glands,

afford instances of physiological fatty degeneration. As a pathological process, however, it depends in some way upon impaired nutrition, is occasioned either by direct interference with the vitality of a tissue, as in inflammation; or by the direct effect of specific poisons, such as alcohol, phosphorus, or the toxins of tubercle and diphtheria; or by depreciation in the quantity or quality of the blood, as in anæmia; or by a diminution in the blood-supply, as in obliterative arteritis and thrombosis.

MUCOID DEGENERATION.—Under this term it is proposed to describe a change in connective-tissue structures characterised by the deposit in its interstices of mucilaginous material (*mucin*). Though somewhat variable in composition, according to their locality, the mucins all belong to a group of substances, known as proteid-carbohydrates, yielding on decomposition a proteid, and a body which reduces Fehling's solution. In this respect mucin is closely allied to cartilage and "amyloid" matter. It swells up in water, is soluble in weak alkaline solutions, and precipitated by acetic acid. It forms the normal secretion of mucous membranes and glands, and the natural matrix of the umbilical cord.

Pathologically, we meet with mucoid formation under apparently very different conditions: for instance, in the inter-vertebral cartilages of old people; in the subcutaneous connective tissue as the result of myxœdema; in new growths, as sarcoma or carcinoma; and in granulation-tissue. Nothing is known as to the conditions which determine this form of degeneration.

The use of the term "colloid," as applied to a special form of degeneration, should now be given up as obsolete. The so-called colloid cancer is but a cancer taking its origin in mucous membrane, and including in its structure cells which produce mucus. The latter may be so abundant as to destroy the cellular contents of the cancer-

alveoli by pressure, so that the growth consists of little else than a fibrous mesh-work permeated by more or less inspissated mucus.

The glairy contents of certain ovarian cysts are closely allied to mucin, but differ from it in not being precipitated by acetic acid.

LARDACEOUS or AMYLOID DEGENERATION.—This form of degeneration is characterised by the deposit in certain tissues of a hyaline or translucent substance belonging (like mucin) to the class of proteid-carbohydrates, but differing in the nature of its carbohydrate moiety, which is more closely allied to that of the matrix of cartilage. Amyloid material exhibits a characteristic mahogany brown colour when treated with a solution of iodine; while with methyl-violet it yields a rosy red colour.

As usually met with, it tends to affect several organs at the same time, occurring most frequently in the spleen, liver, and kidneys. For the most part it appears first in the connective-tissue elements of the smallest arterioles, or just outside the endothelial layer of the capillary walls. As the process extends, the vascular walls gradually lose their structure, become thickened, encroach on the lumen of the vessel, and may exert pressure on adjacent parts—in this way preventing their proper nourishment.

In the liver the terminal branches of the hepatic artery are first affected, the walls of which lose their structure, swelling up and becoming homogeneous. The hepatic cells are compressed, and frequently atrophy and degenerate. In the kidneys the change begins in the vessels of the glomeruli. In the spleen and lymphatic glands, on the other hand, it generally begins in the lymphoid trabeculæ. But wherever the process begins, waxy-looking spots and patches are formed, the exact appearance of which differs according to circumstances. The affected organ or part gradually enlarges, and its consistency

becomes much firmer. In an early stage the change can only be recognised with the microscope; but, later on, it may be readily recognised by the naked eye, assisted by the use of reagents.

The pathological conditions of its occurrence are not known. Clinically, however, it is always associated with grave cachexiæ, especially in connection with chronic tuberculosis, syphilis, long-standing suppuration, and dysentery.

CALCIFICATION, or CALCAREOUS DEGENERATION.—Calcareous formations varying from mere grit to hard bone-like masses or flakes are of common occurrence in old age and in dead or dying tissues. They are composed of lime and magnesian salts, and chemically there is little to distinguish it from the physiological process of ossification, but there the resemblance ends. Calcification, unlike ossification, is a retrograde process, associated with no organisation of any kind, and no special structure.

It occurs most commonly as the result of old inflammation or fatty degeneration. Thus it is frequent in the walls of old phthisical cavities, in atheroma of the aorta, and in the capsules of *trichina spiralis*. When arising from gradual impairment of nutrition, as in old age, extravascular tissues, such as cartilage and elastic tissues, are apt to be first attacked.

The details of its causation are obscure; but in many cases it would appear to be due to precipitation of calcareous matter from the lymph, by acid products of decaying tissue. It may be added that the chemical principles which underlie the formation of calcareous concretions in the ducts of various glands are closely analogous to those which occur in calcareous degeneration, although the remote factors are, of course, quite different.

FATTY INFILTRATION.

When normal adipose tissue encroaches upon muscular tissue, it interferes with the nutrition of the muscular fibres, and causes atrophy with impairment of function. Instances of this are met with in fatty infiltration of the heart and in pseudo-hypertrophic paralysis; also, when fat accumulates in the hepatic cells in abnormal quantity, it interferes with their proper functions, and constitutes a disease. As in adipose tissue, the fat in the hepatic cells appears in the form of large globules, which tend to run together.

Etiologically, it results from a too liberal supply of nutriment, especially of fat and carbohydrates, such as occurs in those who indulge in abundance of rich food and malt liquors; especially when combined with insufficient exercise. Fatty infiltration of the liver also occurs as a result of deficient oxidation, such as may be met with in chronic disease of the lungs. The cause of pseudo-hypertrophic paralysis is very obscure.

NECROSIS.

NECROSIS, or local tissue-death, has already been touched upon in connection with inflammation, infarction, and degeneration; but it seems desirable to bring together its principal varieties into one section, for the purpose of brief description. It will be obvious that, looking at the process from a general point of view, the appearances presented will vary not only according to the mode of origin, but also to the rapidity of development, and the extent of the area affected. For the present purpose it is unnecessary to give detailed consideration to those

cases of slowly progressive decay and ultimate death of tissue resulting from local failure of nutrition, which are sometimes spoken of as *necro-biosis*; and reference will only be made to a few fairly well-defined types of special nature.

Coagulation necrosis.—This is best seen in infarcts. It must not be confused with coagulation of extravasated blood; and, as a matter of fact, the process is especially typical in white infarcts without hæmorrhage. The tissue elements in the affected become pale, lose their structural features, and become merged together to form a solid homogeneous mass suggestive of coagulation. It is not necessarily a fibrinous coagulation, like that of blood. More probably it represents coagulation of another type, though it may respond in some degree to Weigert's fibrin stain.

Allusion has already been made to softening taking place in the interior of old thrombi and hæmorrhages. Such softening represents a special type of necrosis, known as *colliquative necrosis*. It occurs under a variety of circumstances, and is due to self-digestion, set up by enzymes liberated in the process of proteid disintegration. In its more typical form it is quite independent of putrefaction or micro-organisms of any kind; but a closely analogous form of necrotic softening may occur from enzymes yielded by certain bacteria.

Another form of necrosis is termed *caseation*. It is essentially a sequel of fatty degeneration. The elements of the affected part break up into a structureless mass of fatty debris, of the colour and consistence of soft cheese—hence its name. It is apt to occur in the central parts of cellular structures of low vitality, and poorly supplied with blood, such as tubercles and syphilitic gummata. Under certain conditions pus may become inspissated and form cheesy matter.

Lastly, as the result of the plugging of a large artery, acute local infection, or severe injury, a large mass of tissue may mortify, and form what is called *gangrene*. Gangrene may be *dry* or *moist*, according to the amount of fluid in the part.

FEVER.

Fever is a pathological process, of which the most characteristic feature is an undue elevation of temperature (*pyrexia*). The average temperature of the human body is 98·4° F. (36·8° C.), and in health only varies within very narrow limits. Of these variations the most important is that termed the “diurnal variation,” which represents the interval between an early morning minimum and an evening maximum. It rarely exceeds 1° F. Other slight variations also occur, due to the ingestion of food, external temperature, physical exercise, etc., but are clinically unimportant. The increased heat of the body may be evident to the touch, but can only be correctly estimated by the thermometer. The increase of temperature varies from a slight rise to 108° or even 110°. Below 101° we speak of *pyrexia* as slight, above 104° as high, and over 106° as *hyperpyrexia*.

Associated with *pyrexia*, other phenomena are more or less constantly observed. *Emaciation* is a constant and cardinal feature, to an extent which is directly proportionate to the intensity and duration of the fever. The *skin* is generally hot, harsh, dry, and pungent, but is sometimes bathed in profuse perspiration. Headache, restlessness, insomnia, and delirium are common *nervous* symptoms. The *chill* or rigor which so frequently ushers in the febrile state is also a nervous phenomenon of a subjective kind, and is due to spasmodic contraction of the cutaneous vessels from shock. It is therefore most marked when the onset of fever is abrupt and sudden. Under similar

conditions, *vomiting* not infrequently occurs in children, and with young infants *convulsions* are sometimes met with. The *pulse* and *respirations* are usually quickened in proportion to the rise of temperature, while the characters of the pulse are variously modified. *Digestive disturbance* is indicated by a dry, clammy mouth, a coated tongue, thirst, loss of appetite, nausea or vomiting, and constipation. The *urine* is scanty, high-coloured, of high specific gravity, and very acid; its solid constituents are both relatively and absolutely in excess; urea and uric acid are largely increased, while the chlorides are usually diminished.

COURSE AND TERMINATION.—The course of fever varies widely according to its cause. In specific fevers it is generally possible to recognise three stages:—(1) the invasion or onset; (2) the fastigium, or fully developed condition; and (3) the defervescence. Fever terminates in death or recovery. In the event of recovery the temperature subsides suddenly by *crisis*, or gradually by *lysis*.

VARIETIES OF TYPE.—Special terms are employed to denote varieties according to the course and character of the temperature; thus, it is said to be *continuous* when the pyrexia persists for some time without marked variations; *remittent*, when between consecutive accessions of pyrexia the temperature approaches, but does not reach the normal level; *intermittent*, when between consecutive accessions of pyrexia the temperature falls to the normal level; *relapsing*, when paroxysms of pyrexia of considerable duration are separated from each other by periods of complete apyrexia lasting for one or more days.

Asthenic or *adynamic* fever is characterised by a comparatively slight rise of temperature, with symptoms of great weakness and prostration.

A fever is said to be *malignant* when it is exceedingly severe, or of a low and exceptionally fatal type.

The "*typhoid*" state, so-called because of its frequent occurrence in bad cases of typhoid, is by no means confined to this affection. It denotes a low febrile condition which is especially characterised by a tendency to the accumulation of sordes about the lips and tongue; a dry brown tongue; great frequency, feebleness, and sometimes irregularity of the pulse; a liability to passive congestion of dependent parts; and low muttering delirium ending in coma. It is supposed to be due to defective elimination, and consequent accumulation in the blood, of the effete products of tissue-change.

Hectic fever mainly occurs in connection with persistent purulent conditions, as that met with in caries of bone, phthisis, and the like. The pyrexia is of an intermittent or remittent type, comes on insidiously, and is sometimes of long duration. It is further associated with marked progressive emaciation, circumscribed flushing of the cheek, a soft frequent pulse, profuse perspirations, and irregular action of the bowels. Hectic fever is now generally regarded as a form of septicæmia.

CAUSE AND NATURE OF FEVER.—In this connection it is necessary to recall the main facts relating to body-heat. The primary source of the heat of the body is to be found in the oxidation of the food and the tissues which constitute normal metabolism. Next, heat is being constantly lost from the body by radiation, conduction, skin and lung evaporation, and by the bodily excreta. Both heat-production and heat-loss are largely under the control of nervous centres; and these again are controlled by higher nervous centres in such a manner as to adjust the relation of heat-production to heat-loss, and thus to regulate the mean temperature and maintain it more or less steadily at a point largely independent of the surrounding medium.

The increased temperature which characterises fever must be due either to increased heat-production, or to

diminished heat-loss, or to some disturbance in their normal relation to each other. Now, the thermometer will not help us here, since it only records the balance between heat-production and loss at a particular moment for a particular part. To get at the truth, calorimetric observations are essential. In the main, these show that (a) during the period of invasion there is increased production over and above loss, so that the temperature rises; (b) during the fastigium both production and loss are increased so that the mean temperature remains above normal; and (c) during defervescence, loss of heat is increased, while production is diminished, so that the temperature falls.

Experimental observation has shown that fever may occur from any of the following causes: (1) the multiplication within the body of pathogenic organisms; (2) the presence of their products (*toxins*); (3) inoculation with enzymes or ferments; (4) anything which gives rise to disintegration of the blood—in circulation (*hæmolysis*) or after extravasation; (5) inoculation with sterile extracts of animal and vegetable tissues; (6) certain drugs; and (7) irritation of certain regions of the central nervous system.

Numerous as the above causes are, we may fairly recognise, in all but the last, the operation of a common principle, namely, the introduction into the system of something, in the presence of which and under the direct influence of which, the tissue-elements react, and exhibit a more lively metabolism with increased production of heat. We shall learn in the next section that in a majority of cases (though not in all) this reaction of the tissues is attended with the formation of substances called *antibodies*, charged with the power of neutralising in greater or less degree the disturbing agent. So it comes to this, that fever represents a reaction of a protective nature on the part of

normal tissue. When the body is young, vigorous, or specially excitable, we may expect (other conditions being equal) a lively reaction and high fever in the presence of fever-producing causes. In those of feeble vitality, in the aged and depressed, such reaction is likely to be poor. Of course, it may happen that the fever-agent also possesses other properties which may qualify or give a special character to the fever which follows—characters often of so serious a nature as to far exceed the importance of mere temperature in relation to prognosis. Lastly, the fever-agent may be so virulent as to paralyse the tissue elements out-of-hand, and thus prevent febrile reaction of any kind with any protection which it may afford so that death quickly follows.

Nervous fever stands in quite a different category. Owing to irritation of centres controlling heat-production, metabolism of the tissues is increased, and the temperature rises—possibly to a high grade; but no material fever-agent is present, and there is nothing of the nature of a reaction.

TREATMENT.—The removal of pus, the treatment of malarial fever by quinine, and of acute rheumatism by salicyl compounds are almost the only instances in which it is possible to strike directly at the causes of fever. As a rule we are compelled to rely exclusively upon indirect measures, namely, hygienic precautions, appropriate and sufficient nourishment, encouragement of elimination by skin, bowels, and kidneys, and cooling or antipyretic treatment. A word of caution is necessary to prevent a too sanguine estimate of antipyretic treatment. Pyrexia is after all only a constant feature of fever, it does not constitute its essence. That some of the general phenomena of fever are due to high temperature, and that in hyperpyrexia they are of dominant importance, is not denied; and in such cases antipyretic treatment may do much.

But the difference between the behaviour and gravity of different forms of fever depends upon conditions far too complex to admit of measurement by a thermometer.

RELATIONS OF MICRO-ORGANISMS TO DISEASE.

That micro-organisms or microbes hold a definite causal relation with certain diseases, and constitute the *contagium* by which they are transmitted from one person to another, is now an established fact. The organisms in question may be either animal or vegetable in nature. The former belong to the order of protozoa ; the latter to the order of Fungi—divisible into two groups :—(a) Schizomycetes or bacteria, and (b) Hyphomycetes or moulds.

Pathogenic PROTOZOA are the causes of many important diseases, especially those which occur in hot countries. Syphilis is the only disease certainly due to a protozoan which occurs at all commonly in temperate climates. As a class, these organisms are larger and more complex in structure than bacteria ; they have usually a nucleus, sometimes two, and may have large flagella or other organs of locomotion. In many cases they pass through a complicated series of changes in their life-history, and their reproduction is effected by a remarkable sexual process. In many cases, too, and this is especially important in connection with the preventive treatment of these diseases, some part of the life-cycle is passed in one or other of the lower animals (mosquitoes, flies, ticks, etc.), and efforts to stamp out these diseases are directed to the extermination of these intermediate hosts. Thus, a particular species of mosquito is the intermediate host of the protozoa which cause malaria ; and one of the methods adopted for ridding a district of malaria consists in stamping out these mosquitoes from the affected area. Some protozoic

diseases, such as that form of dysentery which is due to amœbæ, are mainly or entirely localised to one part of the body; but most of them are general, the organisms passing into the blood, and affecting all parts of the body. In most cases they tend to run a prolonged course, and immunity is not easily established.

BACTERIA are minute unicellular organisms, which contain no chlorophyll, and are non-nucleated. They may be classified according to their shape, viz. *micrococci*—more or less spherical, and distributed either singly, in pairs (*diplococci*), in clusters (*staphylococci*), in strings (*streptococci*), or in cubical groups (*sarcinæ*); *bacilli*—rod-shaped, but differing much in length and thickness; *comma-bacilli*—also rod-shaped, but curved upon themselves, and, when arranged in rows, assuming a more or less spiral form (*spirilla*). All bacteria multiply by subdivision, and in some species this is associated with the formation of seed-like spores. Their growth and development demand special nutritive material, moisture, and warmth. They are destroyed by exposure to moist heat of 212° F. in a few minutes, and also by certain chemical substances, such as mercuric perchloride, iodine, and carbolic acid; but the spores are far more refractory to destructive influences than the bacteria themselves, a fact of much importance in connection with practical disinfection.

Microbes are found in great abundance almost everywhere, and enormous numbers must therefore be introduced into the body with the food we eat, the water we drink, and the air we breathe. For the most part these organisms are of a fermentative, or of a putrefactive nature (*saprophytes*). Under ordinary circumstances of health the blood and the tissues show no trace of them. Most are destroyed by the hydrochloric acid of the gastric juice, while some pass through the body unaltered. By virtue of some special feature attaching to living blood and tissues,

saprophytes cannot, under ordinary circumstances, maintain their existence within the healthy body. Nevertheless, on dead tissues that are still connected with the body, on shed and partially shed epithelium, on organic accumulations about the teeth, on the organic contents of the alimentary canal, on secretions, and on structures more or less devitalised by inflammation or disease, they find a congenial soil, and forthwith set up the processes—putrefactive or otherwise—which are peculiar to them. Putrefactive decomposition is always attended by the formation of poisonous substances, which differ according to the organism which produces them. These poisons, when absorbed into the system, cause more or less severe constitutional disturbance, due to chemical blood-poisoning (*sapræmia*). Though quite distinct from true infective processes, *sapræmia* has an important influence upon the behaviour and character of any infective disease with which it may be incidentally associated.

Pathogenic bacteria are those which, by growing and multiplying within the body, set up infective disease. The so-called germ theory is based not only upon the association of each infective disease with some specific organism, but upon the experimental causation of the disease in question by the inoculation, after repeated artificial cultivation outside the body, of the special organism originally derived from a similar case. This rigid proof fails in a few cases, such, for instance, as in leprosy; but the evidence with regard to others is more or less complete, and the analogy between all infective diseases is so close, that we are justified in accepting this theory as essentially true.

The mode in which pathogenic microbes enter the body varies considerably. In some cases they do so by accidental inoculation through some breach of an external or internal surface, as in pyæmia, erysipelas, tetanus,

hydrophobia, and glanders. In others they enter by the alimentary canal, as in typhoid fever and cholera; while in a third group by the respiratory passages, as in tuberculosis, diphtheria, and some of the exanthemata. Again, while the contagium in some cases may be carried for a considerable distance in the air, in others it can only be conveyed by direct or indirect contact with the affected person.

The entrance of the organism is followed by a latent period of *incubation* unaccompanied with obvious disturbance, which varies in duration, but is approximately constant for each disease.

The period of incubation is occupied by the multiplication of the micro-organism, until *toxins* (poisonous products) are formed in sufficient quantity to bring about the morbid condition peculiar to the organism in question. Though the clinical phenomena of all infective disease depend mainly on the directly poisonous effects of toxins, the latter do not always get access to the system under the same conditions. In some cases, the development of microbes is confined to a local surface, their toxins alone being absorbed. In others, the microbes themselves enter the body, and circulate with the blood, forming their toxins *en route*. Yet again, as the result of differences in the virulence of the same microbe under various conditions, or in the resistance of the tissues of different animals, or of the same tissue under different conditions, the distribution of the same kind of microbe may be at one time local, and at another time general.

It is a familiar fact that we meet with *varying degrees of susceptibility* to a given infective disease. Some take it readily on exposure to infection; some less readily; while others enjoy complete immunity. If we examine the matter more closely we find that these differences in behaviour depend in part upon factors relating to the

micro-organism, and in part upon factors relating to the individual.

With regard to the micro-organism, much depends on its virulence, the dose received, and the part of the body into which it is received. Again, different results follow according to whether it is introduced into the system in a pure state or in association with other microbes.

With regard to the individual, we find that *susceptibility to infection* varies greatly. It may be *acquired* by anything which tends to depreciate the general health, such as fatigue, inanition, exposure to cold, and loss of blood. Again, it may be *inherited*, of which a notable illustration is afforded by the tendency of tuberculosis to affect several members of the same family. A special susceptibility or predisposition belongs also to early life (excepting the newly born, with whom susceptibility is usually less than in later life).

In like manner, *immunity* may be either *acquired* or *inherited*. It may be acquired in different ways. One of the commonest is illustrated by the protection conferred against a second attack by so many infectious diseases. Such immunity is relative and not absolute: and some forms of infective disease, so far from affording protection, seem rather to increase the liability to recurrence, *e.g.* erysipelas. Immunity may also be acquired in some cases by inoculation with an artificially attenuated culture, or with very minute doses of a virulent culture, or with a vaccine, *i.e.* a sterilised culture of micro-organisms peculiar to the infective disease from which protection is sought (*active immunity*). Another mode of conferring immunity, of especial interest in connection with the treatment of certain infectious diseases, rests upon the fact that when an animal has been immunised against an infective disease by one of the foregoing methods, the blood-serum of that animal, when inoculated into another, confers protection

against that disease (*passive immunity*). Such inoculation is not merely preventive, but if carried out within a short time after the disease has actually broken out, further manifestations are often checked, and recovery takes place. This method of treatment, known as "serum-treatment," is already in general use for diphtheria, with most satisfactory results: and has been extended to the treatment of other specific diseases with varying success.

Strictly speaking, immunity can only be regarded as inherited, when there is a direct transmission to the ovum of an immunity possessed by either parent. Racial immunity, as illustrated by the insusceptibility of negroes to certain tropical diseases, and that enjoyed by some animals with regard to infective diseases which others readily take, is a form of inherited immunity.

The exact *mechanism of immunity* is not yet fully known, but several important facts are fully ascertained. (1) The leucocytes play a part of great importance, ingesting and digesting living bacteria, and thus destroying them (*phagocytosis*). This process is greatly facilitated by the presence of fresh serum which contains substances termed "opsonins," which combine with the bacteria and render them liable to the attack of the leucocytes. The serum from a patient suffering from a given disease often contains less opsonin to the organism which causes the disease than does a normal blood, whereas an immune person may have more. (2) The presence of bacteria in the body gives rise to the production in the blood of protective substances, or *antibodies*. For example, the serum of a person suffering from typhoid fever contains *agglutinin*, which acts upon typhoid bacilli, paralysing them and causing them to collect together into clumps. This is made use of in the *Widal's reaction*, used in the diagnosis of typhoid fever. Other antibodies (*antitoxins*) neutralise the toxins which are formed by the bacteria; whilst yet

others (*bacteriolysins*) dissolve them. It is probable that the phagocytic activity of the leucocytes in conjunction with opsonin is the main factor in the defence of the body, the other antibodies being merely additional safeguards.

HYPHOMYCETES or **MOULDS** consist of cells multiplying by fission so as to form linear threads which interlace with each other and constitute a *mycelium*. In some species (*oidia*) the terminal threads produce oblong or spherical cells which ultimately become free (*conidia* or *spores*). The *oidia* are of importance as the specific organisms of ringworm (*trichophyton tonsurans*), of favus (*achorion Schönleini*) and of tinea versicolor (*microsporon furfur*). The fungus of thrush, formerly described as an *oidium*, is now regarded as a species of *torula* or yeast fungus (*saccharomyces mycoderma*). The *oidia* only affect the skin, producing a local disturbance of nutrition which is unaccompanied by any constitutional symptoms.

SYNCOPE, SHOCK, COLLAPSE.

The above terms are in common use to express different aspects of functional depression; but at the same time it is difficult to differentiate them strictly, either from a clinical or pathological standpoint.

Syncope implies more or less unconsciousness and suspension of function, of sudden onset, and usually of brief duration. In this condition we have to deal proximately with cerebral anæmia resulting from vaso-motor disturbance in association with depressed action of the heart.

In *shock*, a closely similar, but more severe, result is induced by sudden disturbance acting upon the central nervous system, such as strong emotion, rapidly developing infection, or cerebral hæmorrhage; or to the reflex effect of strong peripheral impressions, such as severe pain, surgical injury, intestinal strangulation; or to rupture or

perforation of internal organs. There is reason for thinking that besides inhibition of the heart, there is also some general inhibition of nutritive exchange, as between the tissues and the blood.

In *collapse*, the onset of symptoms is more gradual and insidious, and the duration longer than in syncope or shock. In its most typical form it occurs from severe hæmorrhage, persistent vomiting, or excessive diarrhoea. It would appear that such cases depend in some measure upon inspissation of the blood, and consequent failure of the circulation, due to direct loss of fluids from the body. In other cases, collapse may be associated with shock, as after severe burns or abdominal operations. Here there may be an active determination of fluids from the blood and tissues to the affected parts.

The symptoms of each condition are sufficiently alike to be described together. In well-marked cases, the patient is profoundly prostrate, and more or less unconscious. The skin is pale, cold, and moist; and the features are pinched and drawn. The temperature is usually sub-normal. The pulse is rapid, often irregular, and gets progressively weaker till it can no longer be felt at the wrist. Breathing is shallow, and often sighing. The voice is weak and altered in quality; the tongue and mouth are dry; and the secretions generally are diminished.

DEATH.

The circumstances under which death takes place are very complex and variable. Certain typical modes of death may be distinguished with advantage, though in actual experience they may merge more or less into each other.

1. General exhaustion or *asthenia*, which is characterised by increasing weakness and emaciation, with feeble pulse

and breathing. The mind is clear at first, but becomes gradually more dull and languid, and the patient passes quietly away.

2. Failure of the circulation—either suddenly as in *syncope* or *shock*, or more gradually as in *collapse*.

3. Failure of the respiration. This sometimes occurs suddenly by paralysis of the respiratory centre under circumstances not very well understood. It is an occasional cause of death from chloroform and from brain-tumours. Much more often it is gradual (*asphyxia*) from obstruction in the respiratory passages, from breathing air deficient in oxygen, or from breathing some gas (like carbonic oxide) which interferes with the appropriation of oxygen by the hæmoglobin of the blood.

4. General failure of the brain functions (*coma*). It begins with the suspension of the higher nervous faculties, giving rise to progressive insensibility and stupor; while it culminates in a final arrest of function in those centres of the medulla oblongata which preside over respiration and circulation.

SECTION II.

SPECIFIC INFECTIOUS DISEASES.

TYPHUS FEVER.

THERE is little doubt that typhus fever is primarily due to a specific organism, though it has not yet been isolated and identified. At the same time, predisposition is of unusual importance, and is especially created by such factors as overcrowding, filth, and famine. With the removal or abatement of these conditions typhus fever has become a rare disease. It is eminently infectious at close quarters, and especially where several cases are aggregated together. Infectiveness is readily weakened by free supply of fresh air, but the disease may be carried for an indefinite distance by contaminated clothes and other articles. No age is exempt from attack, but second attacks are rare.

MORBID ANATOMY.—The *post-mortem* appearances are characterised by *disorganisation*. The blood is dark, and coagulates imperfectly; the muscles are softened and degenerated; and many of the internal organs become enlarged, hyperæmic, and softened.

SYMPTOMS.—The period of *incubation* is about twelve days, and may be accompanied by slight constitutional disturbance. In a typical case the *invasion* is generally abrupt, and is ushered in with chilliness or rigors, nausea or vomiting, severe headache, and steady rise of temperature. Prostration and depression are present from the outset, in direct proportion to the severity of the case;

and the general character of the disease is that of acute blood-poisoning of a low type. Nervous symptoms are prominent, and come on early. Almost from the first there is marked restlessness with disturbed sleep at night. Pains in the back and limbs, giddiness, intolerance of light, and deafness are usually present. More or less delirium is common, and in alcoholic cases is apt to be extremely violent. The expression is dull, the complexion dusky; and the skin emits an unpleasant mouse-like odour. The tongue is coated, and trembles when protruded. Thirst, nausea, or vomiting, with almost complete loss of appetite, are frequent symptoms. The bowels are generally confined; but when relaxed the stools are dark-coloured, and quite unlike the pale shreddy evacuations of typhoid fever. Respiration is notably hurried; and the pulse is quick, soft, and compressible. The urine is diminished in amount, and almost always contains albumen. The temperature will be considered separately.

The eruptive stage.—The eruption consists of *maculæ* or “mulberry spots,” usually combined with more or less *subcuticular mottling*. The *maculæ* appear about the fourth or fifth day on the front and back of the trunk—and, if abundant, on the limbs also. The face is not affected. They come out in one crop, and begin to fade in the second week. They consist of irregular round reddish spots, which (when fully developed) are hæmorrhagic, of a dark-red mulberry-juice colour, and do not disappear on pressure. The *mottling* is of a dusky-red colour, and appears as if beneath the surface of the skin.

During this stage the previous symptoms increase in severity. Headache becomes replaced by delirium, which is usually of a low muttering type. It tends to merge into stupor, and the special senses are dulled. The tongue becomes dry, brown, and cracked; and the lips and teeth are encrusted with sordes. Prostration steadily

increases; the pulse rises to 130 or 140, or more, becoming very feeble and fluttering; and the first sound of the heart is apt to be indistinct or even inaudible—indicative of threatening cardiac failure. Respiration is hurried and disturbed. The urine is very scanty, high coloured, and frequently contains albumen. In bad cases it is often retained, or dribbles away involuntarily with the stools. Bed-sores are common.

Stage of decline.—The average duration of typhus fever is from fourteen to twenty-one days. Death is most frequent about the end of the second week, either as the result of profound exhaustion, or of some complication, such as pneumonia. In favourable cases—which are the rule—the fever ends by *crisis*, about the thirteenth day. The patient falls into a good sleep, from which he awakes greatly relieved. The tongue moistens, the mind clears, the appetite returns, and recovery often sets in with wonderful rapidity.

Course of the temperature.—There is a rapid rise of temperature from the first, reaching a maximum of 103° to 105° by the fourth or fifth day, with only slight morning remissions. A more marked remission is often observed about the sixth day; but the temperature soon rises again and is maintained in favourable cases till the final fall, which is sudden and complete. In fatal cases, however, death may be preceded by a rise to 108° or 109° .

The *complications* of typhus are due, for the most part, to the prostration, disorganisation, and toxæmia which characterise this fever. Cardiac softening and degeneration lead to extreme feebleness of the circulation, thus creating a liability to hypostatic congestion of the lungs, and broncho-pneumonia. More rarely, local infective mischief gives rise to parotid bubo, abscesses, gangrene, or renal disease.

The main *diagnostic* features are—sudden onset, early

prostration, early appearance of nervous symptoms of a low type, and the "mulberry rash." In sporadic cases the diagnosis may be very difficult, till the rash is well out.

As to *prognosis*—advanced age, previous intemperance, great feebleness or extreme rapidity of the pulse, hiccough, suppression of the urine, pronounced delirium alternating with stupor, are very unfavourable. The mortality varies from ten to twenty per cent.

TREATMENT.—The patient should have complete rest in bed, in a well-ventilated airy room. The food should be liquid and nutritious; and water should be freely given. Alcohol is certainly useful in most adult cases, especially in the later stages of the disease. The mouth should be frequently and carefully cleansed. The urine should be drawn if necessary; and precautions should be taken to prevent bed-sores. Medicinally, the mineral acids and quinine are chiefly recommended. The *nervous symptoms* are best met by tepid spongings of the body, and the application of ice to the head: *violent delirium with excitement*—by wet-packing, sulphonal, paraldehyde, or hyoscine hydrobromate; *low muttering delirium* and the "*typhoid*" state—by stimulants (opium is contra-indicated); *stupor*—by strong coffee, and by encouragement of elimination by the skin and bowels.

TYPHOID FEVER—ENTERIC FEVER.

Typhoid fever is an acute disease, due to infection by a specific organism, known as the Eberth or "typhoid" bacillus. It is found not only in the blood, fæces, and urine of the patient, but in the lymphoid follicles of the bowels, mesenteric glands, and spleen. It is short, thick, and provided with flagella. It does not form spores. It closely resembles the colon bacillus, and can only be distinguished by its behaviour in culture.

The contagium is perhaps most often conveyed to the body in drinking water contaminated with soakage from privies, cesspools, or drains. Milk may be a vehicle of infection, by the use of contaminated water for cleansing dairy cans. Uncooked articles of food, such as oysters, water-cress, and salads may also communicate the disease, by previous contamination with infected sewage. Again, in hospitals, it may be caused by direct contact with typhoid stools, or by the inhalation of dust from soiled bed-linen. Flies may also carry infection to food from exposed excreta. Lastly, it is important to bear in mind that those who have had typhoid fever may carry about the bacillus in the bowel or its appendages long after convalescence, and so become a possible source of infection. The disease chiefly prevails in the autumn months, and is most common between the ages of ten and twenty-five years.

MORBID ANATOMY.—The most characteristic lesions affect the lymphatic structures of the abdomen:—1. *Inflammation and ulceration of the solitary and agminated follicles (Peyer's patches) of the intestine.* These changes begin and are most extensive in the neighbourhood of the ileo-cæcal valve, becoming less marked on proceeding upwards from this point. The follicles of the cæcum, appendix, and colon are less often involved. The follicles become highly congested, and greatly swollen by infiltration with lymph cells. This infiltration may extend beyond the follicles to the adjacent mucosa. During the second week the infiltrated tissue undergoes necrosis and becomes detached during the third week, either entire or in fragments, leaving an ulcer. The shape, size, and extent of the typhoid ulcer correspond to the amount of necrosis. It is rounded or oval in shape as a rule, but occasionally a large and irregular ulcerated surface is exposed by the fusion of adjacent ulcers. The floor is formed by the sub-

mucous, muscular, or peritoneal coats of the bowel according to circumstances. Its edges are thin, soft, and undermined. Usually, during the fourth week, cicatrisation takes place from the edges inwards, finally resulting in a depressed and slightly pigmented scar. *Perforation* of the intestine may occur directly from extension of the ulcer in depth, or from detachment of a slough involving the whole thickness of the gut, or from laceration. *Hæmorrhage* from the bowel is also not an uncommon event. Early hæmorrhage (occurring during the first ten or twelve days) is generally slight, and depends upon vascular rupture from extreme congestion of the intestinal mucous lining. Late hæmorrhage (most frequent during the third week) is usually abundant, and results from erosion of a larger vessel in the floor of the ulcer. 2. The *mesenteric glands* become infiltrated, enlarged, and softened. 3. Similar *enlargement and softening of the spleen* is very constant. 4. *Ulceration of the larynx* is occasionally found.

The lungs invariably display hypostatic congestion at their bases, which may pass on to pneumonic consolidation. The heart is often softened, as the result of granular degeneration. More rarely, some of the voluntary muscles, especially the recti abdominis, undergo localised coagulation necrosis (myositis typhosa), leading to rupture of muscular fibres and hæmorrhage on slight provocation.

SYMPTOMS.—The period of *incubation* is difficult to determine, but is certainly under a fortnight, as a rule.

The *attack* usually begins insidiously. The patient is feverish, and at first complains of little more than frontal headache, abdominal discomfort, general malaise and aching pains in the limbs. Epistaxis is also not uncommon.

At first the symptoms are not, as a rule, so severe as to compel the patient to take to his bed; but the general condition soon becomes more characteristic. The countenance is pale, though the cheeks may be flushed; the

skin is usually dry, but sometimes bathed in sweat; the frequency of the pulse is generally much less than during the invasion of other fevers—often under 100, when the temperature is above 104° ; the appetite is lost, and the thirst is marked. At this stage, the nervous disturbance is usually limited to headache and restlessness. The tongue is usually coated with a coarse white fur, which thins off towards the tip and edges, and is often cracked transversely. By the end of the first week, the following abdominal signs may be usually observed:—*Pain and tenderness*, especially over the right iliac fossa; *fullness and tumidity*; *enlargement of the spleen*; and *diarrhœa*. The behaviour of the bowels is not constant. They may be relaxed from the outset, the stools—which are at first fæcal—gradually assuming the appearance described below. But, more often, the bowels are confined in the first instance, becoming relaxed later on; while sometimes (especially in children) they are confined throughout. Typical typhoid stools are of thin consistence and pale yellowish colour, resembling pea-soup, shreddy, of an offensive odour, and of alkaline reaction.

The *eruption* first appears from the seventh to the tenth day, upon the abdomen, chest, and back; it comes out in successive crops, each spot lasting for three or four days. The number of spots varies much. The average duration of the rash as a whole is about a fortnight. The spots are roundish, of the size of a mustard seed, slightly elevated, well defined, rose-coloured, and disappear completely on pressure, and after death.

During the *second week* the countenance becomes more flushed, and the patient rapidly emaciates. The frontal headache disappears, he becomes drowsy, and is often more or less apathetic, with slight nocturnal delirium. All muscular movements are feeble and uncertain. The tongue becomes drier and cracked in the centre, and

trembles when protruded : the abdominal symptoms increase, and hæmorrhage from the bowels may occur. The pulse becomes more frequent and feeble. There is usually some little cough and expectoration ; and the physical signs of hypostatic congestion may always be detected over the posterior bases of the lungs.

In the *third week* the patient is at his worst. Dull, tremulous, and prostrate, he lies helplessly on his back ; the pulse is rapid, compressible, and dicrotous ; the tongue is brown and dry, with sordes on the teeth and lips ; and the throat is often sore. The distension of the abdomen increases (tympanitis) ; and, if pronounced, is a bad sign. Diarrhœa may be severe, and it is about this time that hæmorrhage and perforation are most apt to occur. The degree of nervous disturbance varies, but it is usually a prominent feature. Besides prostration and tremor, there may be deafness, and delirium—usually of a low muttering type—occurring by day as well as by night. In very severe cases, the patient is apt to pass into what is called the “typhoid state,” in which delirium merges into coma, with twitching of the wrists (subsultus), and picking at the bed-clothes (carphology). The fæces and urine may be passed involuntarily, and bed-sores are not uncommon.

About the *fourth week* the symptoms usually begin to subside in favourable cases. Defervescence is by *lysis*. The total duration varies considerably ; in mild cases it may be less than four weeks, while in severe cases it may be much longer. The convalescence is slow and liable to interruption from relapse.

In *relapse*, the phenomena above described reappear, usually (but not always) in a milder form, and of shorter duration than the primary attack. There is no constancy as to the duration of the interval, which, in my own cases, has varied from two to eighteen days. Sometimes there is a second or third relapse.

Temperature.—The primary onset of the fever is very characteristic; the temperature rises about two degrees every evening, and falls about one degree every morning for the first four or five days, attaining a maximum towards the end of the first week. During the second week, this maximum is usually maintained, and the fever is of a continued type. From this point, the daily oscillations become exaggerated; being first of a remittent and afterwards of an intermittent type, gradually subsiding, and in average favourable cases reaching the norm some time during the fourth week. Occasionally convalescence is ushered in by a somewhat abrupt fall. In more severe cases the period of exaggerated daily oscillation may be considerably prolonged, with intercurrent irregularities. A fatal termination is sometimes marked by a rapid rise to a high grade, or sometimes by a sudden fall, with collapse. During convalescence the temperature may from time to time exhibit a sudden temporary rise, which must not be confounded with “relapse.”

Blood.—The red cells are progressively diminished in number as the disease progresses. The number of leucocytes is also usually decreased; but any inflammatory complication is liable to lead to abnormal increase. The serum of typhoid blood, when added to a culture of typhoid bacilli, causes the latter to clump together and become motionless (*Widal's reaction*). The reaction is seldom well marked before the second week, but it may continue for a much later period.*

Urine.—During the febrile period, the urine is reduced in quantity, with a sp. gr. of 1.025 and upwards, and considerable increase of urea and uric acid. During defervescence it becomes much more abundant, limpid, and pale, with a low sp. gr. Ehrlich's diazo-reaction* is seldom absent, and is best marked during the second week.

* See Appendix.

It is found, however, in several other specific fevers, and has chiefly a negative value. Slight and temporary albuminuria is common; but it may sometimes be considerable, and accompanied with renal epithelium, blood-cells, and casts, as the result of nephritis.

VARIETIES.—Occasionally the onset is abrupt and attended with marked nervous, gastro-intestinal, or renal disturbance. Or again, the disease begins with acute pneumonia, and does not reveal its true nature until after a week or more. Sometimes the course of the disease is very mild and short, while in other cases it is grave, prolonged, and attended with early nervous prostration. In another form the disease is apparently mild, and the patient goes about his usual duties, until suddenly overtaken by delirium, hæmorrhage, or perforation (*ambulatory typhoid*).

COMPLICATIONS AND SEQUELÆ.—*Hæmorrhage* may occur within the first fortnight; but, in this case, is seldom of much importance, and is probably due to congestion. Later hæmorrhage from ulceration is a more serious affair, and always gives rise to anxiety. Fortunately, it is not common, its frequency being variously estimated at from 4 to 8 per cent. Its amount varies from small clots passed from time to time with the stools, to hæmorrhage so profuse as to be quickly fatal. Under ordinary circumstances it is attended with an abrupt fall of temperature, which may precede the actual discharge of blood *per anum*, a rapid pulse, pallor, and faintness. The fall of temperature is temporary; and if the patient survives, it soon regains its previous level, or may exceed it. The colour of the discharges is darkened in proportion to the delay of the blood in the bowel before evacuation.

Perforation of the bowel occurs in about three per cent. of all cases, and is almost always fatal, as the result of acute peritonitis set up by the escape of the intestinal

contents into the abdominal cavity. Its recognition always requires care, and may be very difficult. The most significant signs are sudden abdominal pain and tenderness (especially when previously absent), shivering, more or less rigidity of the abdominal walls with immobility on breathing, and (in well-marked cases) obliteration of liver-dullness. The temperature may fall sharply, but by no means always. Peritonitis soon develops, and is manifested by vomiting; increased tenderness, resistance, and distension of the abdomen; a rapid thready pulse; an expression of extreme anxiety, and cold extremities; passing on to complete collapse and death within three days at most. In some cases, either as the result of a very small perforation, or because the patient is already more or less comatose, these severe symptoms may be absent, and a marked increase in abdominal rigidity or distension may be the only evidence of what has happened. More rarely, as the result of peritoneal adhesions, recovery may occur, or a local peritoneal abscess may take the place of general peritonitis.

Among other important complications which may occur are the following:—acute pneumonia, ulceration of the larynx (sometimes leading later on to suppurative perichondritis and necrosis of the cartilages), unaccountable recurrent rigors, myocarditis, and wholesale ulceration of the colon.

During the period of convalescence we may meet with falling off of the hair, desquamation of the skin, extreme emaciation, evidence of peripheral neuritis, tenderness of the toes, neuralgic pain in the lower part of the back (typhoid spine), thrombosis of the femoral vein, superficial suppurative osteitis, abscesses, and psychical troubles such as melancholia, dementia, and mania. Pregnant women usually abort, but not always.

The *diagnosis* is often very difficult, especially during the first few days. A rule of considerable service in

practice is this—any acute fever, lasting for a week, without any rash or apparent local mischief, is probably either typhoid or tuberculosis. Acute tuberculosis, influenza, ulcerative endocarditis, certain forms of enteritis, and appendicitis are the diseases which are most apt to be confounded with typhoid.

The *prognosis* is favourable on the whole, but the possibility of death from perforation or hæmorrhage gives an element of uncertainty to every case. Protracted high temperature after the end of the second week, progressive frequency of the pulse beyond 120, weakness of the cardiac first sound, pronounced tympanitis, severe and early delirium, and pneumonia are of serious import.

TREATMENT.—The patient should be kept strictly to bed in a well-ventilated room, and especial care should be taken to prevent soiling the bedclothes with fæcal matter, and to remove any soiled linen as quickly as possible. The stools should be disinfected before being thrown away.

Strict rules as to the *diet* are imperative. Milk alone (diluted with water, barley-water, or soda-water) is best. Two pints (rarely more) should be given in twenty-four hours. Special caution is necessary against its indiscriminate use, for if undigested curd passes on into the bowel it may set up violent irritation. It should be given in divided doses at regular intervals, and drunk in sips—not in a single draught. If not digested in this form, unsweetened condensed milk, peptonised milk, or milk which has been kept for one to two hours at a temperature of 150° F. may be tried. If milk is resented, or variety needed, broths, beef juice, clear soups (fortified by the addition of well-cooked rice or plasmon), meat-jellies, baked or boiled custard, or pulped meat and breadcrumbs may be given. Water and simple cooling drinks should be freely allowed. The mouth should be kept clean, and regularly swabbed

with equal parts of boroglyceride and glycerine. *Alcohol* is not needed in ordinary cases, and rarely before the end of the second week. A progressively frequent pulse, feeble heart-sounds, muttering delirium, inability to take food, and previous alcoholic abuse are the chief indications for it.

In mild cases no medicinal treatment is necessary beyond some simple saline, or a mixture of dilute hydrochloric acid and chloroform water. Under other circumstances there is much evidence in favour of intestinal antiseptic medication, such as β -naphthol, gr. 5, every 4 or 6 hours; or calomel, gr. ij.-iv., every second or third day followed within six hours by a full enema. When the temperature runs high, antipyretic treatment by cold packing, or full baths cooled down from 90° to 75° are of service; and in all cases the patient should be kept as cool as possible with frequent, tepid spongings. Medicinal antipyretics are better avoided.

As to special symptoms:—*Excessive diarrhœa* demands strict scrutiny of the diet; and, if further treatment is needed, the colon should be washed out with a full injection of hot water, followed by an occasional dose of Dover's powder, or an opiate enema. *Constipation* must be dealt with discreetly. Nothing but a moderate dose of castor oil (repeated if necessary) or a simple enema should be employed. *Hæmorrhage* should be treated by absolute rest, cold applications to the abdomen, and the internal administration of acetate of lead with opium. *Perforation*, as already mentioned, is nearly always fatal; but if it is diagnosed early, operative treatment should receive serious consideration, since it may afford at least a possibility of success. *Tympanitis* demands careful regulation of the diet. Thin barley-gruel and weak broths may often be substituted for milk with great advantage; turpentine stupes should be applied to the abdomen; and turpentine in doses of $\mathfrak{m}$ xv.-xx. every four hours, or small and fre-

quently repeated doses of calomel may be given. Hot water enemata are also useful. *Nervous excitement* and *sleeplessness* are best controlled by spongings or wet packing, and the internal administration of bromide of ammonium, or an opiate such as Dover's powder. In delirious cases, a watchful eye should be kept on the bladder.

During *convalescence* the diet should be light, and moderate in quantity. More substantial food should be resumed gradually, and no solid food should be given for at least a week after the temperature has become normal. Light wine or beer is often useful at this stage.

RELAPSING FEVER.

This is a peculiar specific fever, characterised by periodical intermissions and relapses. The conditions under which it arises are much the same as in typhus, but it is not so infectious, and generally occurs in epidemics. It is especially common in seasons of famine. No age is exempt; but it occurs most frequently between fifteen and twenty-five years of age. One attack confers only slight protection from subsequent attacks. Its incubation is probably under nine days, but it varies a good deal. The specific cause of the disease is a motile *spirillum* (*Spirochæta Obermeieri*), which abounds in the blood during the febrile period, but is absent during the intervals of apyrexia. There are no characteristic *anatomical changes*.

SYMPTOMS.—The *invasion* is remarkably sudden, setting in with rigors, frontal headache, giddiness, and marked pains in the back and limbs, with great restlessness and sharp rise of temperature. The most striking symptoms at this stage are—rapid prostration, often accompanied with nausea and vomiting, and sometimes with jaundice;

severe pains in the body and limbs ; and marked tenderness over the liver and spleen, which undergo notable enlargement. The tongue is coated, and later on becomes dry. The bowels are generally costive. Delirium is rare, in spite of the fever and prostration, which reach a maximum from the fourth to the sixth day. The urine is febrile, and often contains albumen and casts. There is no eruption.

The general condition now appears grave indeed ; but about the fifth to seventh day a profuse perspiration breaks out, the pulse falls to its normal standard, the fever disappears by *crisis*, the tongue gets moist, and the appetite quickly returns. The patient feels much better, and looks forward to a speedy and complete recovery ; but in this he is disappointed. Generally, in about seven days (twelve to fourteen from the first onset) there is a *sudden relapse*, with the reappearance of all the former symptoms. The intensity of the relapse is generally less than that of the first attack. The relapse continues from three to five days, when a *second crisis* occurs, usually bringing the disease to a close ; but a second or even a third relapse may occur. The debility which results is considerable, and the convalescence is slow.

The *temperature* rises rapidly, and reaches a maximum of 104° to 105° on the fourth or fifth day, and is maintained with slight morning remissions until the critical fall. During the relapse the temperature does not usually rise so high as before, and reaches its maximum earlier.

In the ordinary type of the disease the mortality is seldom above five per cent. In fatal cases death most often occurs during or after the first relapse, sometimes from exhaustion or collapse, but usually from some complication, such as pneumonia, severe diarrhœa, or hæmorrhages. Pregnant women always abort.

TREATMENT.—No specific form of treatment is known.

At the outset, gentle aperients with cooling drinks, a light nutritious diet, and perfect rest are required. Tepid sponging often gives great relief. Opium is said to greatly relieve the headache, muscular pains, and insomnia. It may be advantageously combined with atropine, and given hypodermically. Quinine has no influence in preventing the relapse.

SCARLET FEVER.

Scarlet fever is undoubtedly a specific disease, but all attempts to relate it to a special organism have so far failed. It is most prevalent in this country in the autumn months. It is eminently infectious; and infectivity specially attaches to discharges from the throat or nose, and scales shed from the skin during the period of desquamation. The disease is also readily spread indirectly by contaminated clothing, books, etc., and sometimes by the milk supply. The period of quarantine which is usually enforced is six weeks from the date of eruption, and, for healthy persons, a week from exposure to infection. A second attack is rare. The disease is much more frequent among children than adults. There are no characteristic anatomical appearances.

SYMPTOMS.—The period of *incubation* is short—usually two or three days. The *invasion* is very sudden, and is ushered in by chilliness and vomiting, or possibly convulsions—in infants. The temperature rises rapidly to a high grade, and is accompanied with the usual febrile symptoms. The pulse is accelerated out of all proportion to the temperature. The tongue is more or less uniformly coated at first; and some soreness of the throat is often complained of. Headache and slight nocturnal delirium are of common occurrence.

The *eruption* varies considerably in intensity. It first

appears about the neck and shoulders on the second day, and extends thence over the trunk and limbs in the course of twenty-four hours. The face is simply flushed, except around the mouth, which is pale. The rash consists at first of bright red punctiform spots, which soon fuse together, till the skin becomes of a bright scarlet hue, readily dispelled by pressure. It reaches its height in two or three days, and then begins to fade. It disappears altogether by the beginning of the second week; and in mild cases still earlier.

The amount of throat trouble varies considerably. In some cases there is little or no soreness, and nothing can be seen but diffused redness and tumidity of the mucous membrane. In other cases there is much soreness, the tonsils are enlarged and inflamed, and the glands at the angle of the jaw are enlarged and tender. The temperature attains its maximum with the rash, and falls to normal as the latter disappears, accompanied by a simultaneous fall in the frequency of the pulse. During the eruptive period, the tongue rapidly cleans from before backwards, and becomes of a bright red colour, with large prominent papillæ (*strawberry tongue*). The bowels are often relaxed at the outset; but later, constipation is the rule. The urine is of febrile type, and may contain a little albumen. In simple cases the headache and nocturnal delirium disappear as the rash comes out.

Desquamation, or peeling of the skin, begins soon after the rash has faded, and is first likely to be noted on the lobes of the ear, or on the neck, extending thence downward over the trunk and limbs. Where the skin is thin, the scales are small and branny; where it is thick, as on the hands and feet, it may come away in large flakes. Its duration varies greatly according to the treatment adopted, but it is rarely completed within six weeks from the date of eruption. The symptoms meanwhile completely sub-

side ; the urine becomes more abundant, and may contain a quantity of renal and vesical epithelium. The temperature is often sub-normal during the earlier part of this stage.

VARIETIES.—1. *Scarlatina anginosa*. In this variety we meet with severe ulcerative inflammation of the tonsils, which may extend to the pharynx, nares, or larynx, and is attended with irritating discharge from the nose. The glands in the neck are greatly enlarged, being sometimes accompanied with widespread infiltration of the neck, which may suppurate and form an abscess. Deep sloughing of the throat structures sometimes occurs, which may lead to fatal hæmorrhage. The rash is usually well marked, but apt to be dusky in colour, and blotchy. Patchy septic rashes are often seen, especially about the elbows and knees. Otitis almost always occurs, and arthritis is fairly common. The fever is of an irregular septic type ; and, coupled with the extreme difficulty of adequate nourishment, leads to profound exhaustion. It is needless to say, that such cases more often end fatally. 2. *Scarlatina maligna* may be characterised from the first by grave nervous symptoms, such as great prostration, restlessness, and delirium, which quickly prove fatal, with coma or convulsions. More often, however, it is less rapid, and the earlier symptoms are those of progressive heart-failure. In either case it is nearly always fatal. 3. Occasionally, instead of subsiding with the rash, fever of a remittent type may continue for two or three weeks without obvious lesions to account for it, and may end fatally by exhaustion. 4. When scarlet fever attacks a woman at her confinement, it is apt to lead to grave septic trouble—e.g. puerperal peritonitis.

Complications are of frequent occurrence. Of these the most important is *acute nephritis*, which is apt to supervene about the end of the third week from the beginning of the

fever. It appears to be quite independent of the severity of the primary fever. It is characterised by albuminuria and sometimes by dropsy. The urine is dark coloured or smoky from the presence of blood. Recovery takes place as a rule, but a fatal result is not uncommon, and in some cases albuminuria persists for weeks or months after all other symptoms have disappeared. *Purulent inflammation of the middle ear*, extending from the throat along the Eustachian tube, followed by perforation of the tympanic membrane, is a still more common occurrence. Amongst other complications the following may be mentioned: abscesses in the neck, arthritis (especially of the wrists and hands), empyema, and broncho-pneumonia.

The *diagnosis* of scarlet fever from other eruptive fevers will be given further on. It should not be forgotten that scarlatina-like eruptions of septic origin are sometimes associated with the early stage of small-pox, and with surgical wounds; and considerable care is necessary in order to interpret them aright. Erythematous enema and drug rashes should also be remembered in this connection.

The *prognosis* of simple uncomplicated scarlet fever is generally favourable; and the gravity of any particular case is usually in direct proportion to the number and importance of the complications.

TREATMENT.—The patient should be isolated in a well-ventilated room, from which all superfluous furniture should be at once removed. The body should be washed daily in bed, and followed by inunction with equal parts of olive and eucalyptus oils. If the rash comes out badly, warm packing is of service; and, in all cases where the fever runs high, frequent sponging is very desirable. An aperient should be given at the outset; and if much restlessness and excitement, some simple hypnotic may be required.

As to diet, milk or milk-food is to be preferred. Where swallowing is difficult, junket or gelatinised milk in small pieces is often taken more easily ; or nasal feeding may be necessary. Alcoholic stimulants are not needed in ordinary cases ; but in bad septic forms they are of great value, if not essential. Watery drinks should be freely allowed ; and, when sufficient fluid cannot be taken by the mouth, subcutaneous or rectal injections of warm saline fluid should be given.

The management of the throat is very important. The mouth should be kept scrupulously clean. While in ordinary cases a simple borax mouth-wash, or swabbing with equal parts of boroglyceride and glycerine may suffice, yet when the throat-mischief is severe, nasal irrigation is essential, combined with frequent spraying of the throat and nose with solution of mercury perchloride (1 in 2,000). Great relief to the throat may sometimes be given by frequent sucking of small pieces of ice, with external application of hot fomentations. In *otitis*, the pain may be relieved by hot fomentations, syringing the ear with hot water, and instillation of a few drops of laudanum, five per cent. solution of cocaine, and glycerine (equal parts of each). When pus has formed, it is better practice to puncture the tympanic membrane carefully by incision than to wait for spontaneous perforation, and the ear should be washed out frequently with warm antiseptic lotion. During desquamation, daily bathing, followed by inunction of the whole surface with carbolic oil—two per cent.—should be employed. In the septic variety, nourishing food, stimulants, and strychnia are specially needed.

MEASLES--MORBILLI.

Measles is an acute specific disease, especially common amongst children, and dependent upon a specific poison

which is contained in the catarrhal discharges from the patient, but not yet identified. It may also be communicated by the clothing, etc. ; but the infectiveness of measles is much less persistent than that of scarlet fever. Infectivity soon declines, and probably disappears with the cessation of the catarrhal symptoms. An interval of a fortnight after exposure to infection is ample to allow before intercourse with others is permitted, provided the exposed person shows no catarrhal signs whatever. A second attack is rare. There are no characteristic anatomical changes.

SYMPTOMS.—The period of *incubation* is about ten days. Though *invasion* is rapid, it is less sudden than that of scarlet fever, and therefore initial shivering, vomiting, and convulsions are less frequent. The temperature soon rises to 102° or 103°, or higher in grave cases. The early symptoms are those of a bad “cold in the head.” The eyes are suffused and watery ; the eyelids red, with some intolerance of light and frontal headache. Epistaxis is not uncommon. The catarrh extends into the throat, air-passages, and alimentary canal, causing sore-throat, hoarseness, cough, expectoration, slight difficulty of breathing, and wheezing sounds in the chest ; loss of appetite, gastric discomfort, with constipation or diarrhœa. On the second or third day, transient improvement, with a remission of temperature, commonly occurs. During this period—often as early as the second day—very characteristic spots are found on the inner surface of the cheeks (*Koplik's spots*). They are usually first seen opposite the first molar teeth, as minute bluish-white specks, resting on a bright red base. In the course of a few days they become more numerous, and cover a wider area. As they occur in no other disease, they have great diagnostic importance. Blotchy erythematous rashes on the skin are also sometimes met with during this period, but they subside before the proper eruption appears.

The *eruption* appears on the fourth day, first on the forehead, thence spreading to the rest of the body. It consists of irregular raised spots or patches of a dusky-red colour, on a background of pale skin. The skin around the mouth is not clear as in scarlet fever, but, on the contrary, is invaded by the eruption. At first the rash disappears completely on pressure; but when it has been out for some time, and for a while after it has faded, there is usually some staining of the skin. It lasts altogether four or five days, and is followed by an insignificant powdery desquamation.

The catarrhal symptoms persist, and still give the affection its chief characteristic; but the mucous secretions now become altered, being no longer clear and watery, but turbid, thick, and yellowish. The margins of the eyelids and nose are red and tumid, while the tongue is usually moist, thickly coated, and often "patchy" in appearance. There may also be some vomiting and diarrhœa at this stage. The temperature, as already pointed out, commonly remits just before the rash appears; but then rises again; reaches a maximum with the rash; and usually subsides rather quickly about the sixth or seventh day in favourable cases. The urine is febrile.

VARIETIES.—Cases of measles differ considerably from each other according to circumstances. For instance, the catarrhal trouble in one case falls chiefly upon the respiratory passages; in another, upon the alimentary canal; while in a third, nervous symptoms are especially prominent. Again, every degree of severity is met with, from the simplest cases which all recover, to those malignant and hæmorrhagic forms which are invariably fatal.

The most important *complications* are: laryngitis, bronchitis, catarrhal pneumonia, pulmonary collapse, inflammations of the eye, ear, or nose, and gangrene of the mouth or vulva.

The *diagnosis* will be afterwards considered. The *prognosis* is favourable on the whole in normal previously healthy children; but it should be remembered that the complications are numerous, of frequent occurrence, and apt to lead to many forms of bodily defect, especially in weakly children. Again, malignant cases are more often fatal.

No special *treatment* is needed in simple cases, beyond the relief of symptoms. But it is wise to carefully cleanse the eyes, nostrils, and throat from secretions by means of mild antiseptic washes. Nervous symptoms are best combated by tepid packs or baths.

Any threatening of the "typhoid state" should be met at once by the free use of stimulants and nourishing food. If there be any hereditary predisposition to tubercle, measles is liable to become the starting point of actual tuberculous disease. Accordingly, it is very desirable that the patient should be placed during convalescence under tonic and invigorating conditions of life, especially in reference to good food and fresh air.

EPIDEMIC ROSEOLA—RUBELLA.

Epidemic roseola, Rubella, or German measles, is an infectious eruptive fever, of more common occurrence at a later age than measles. Its infectivity is of quite short duration, and relatively mild. A quarantine period of three weeks is sufficient for those exposed to infection. Second attacks seldom or never occur. The incubation period is about eighteen days.

Slight febrile symptoms usually precede the appearance of the eruption for about one day. The rash first appears on the nose and cheeks in the form of small round or oval red spots, slightly elevated, disappearing on pressure, which rapidly extend over the body, and may coalesce as they

increase in size, forming patches or large areas of uniform redness. The eruption usually fades within two days, and is followed by slight desquamation. The eyes are red, but there is little or no coryza; the throat is almost always sore, and the glands in the neck along the posterior border of the sterno-mastoid more or less swollen and tender. It is worth noting that such glandular swelling may usually be found (if looked for) some two or three days before the rash appears, thus often providing the earliest indication of the disease. The amount of general disturbance is very trivial indeed, as a rule, and the duration rarely extends beyond four or five days. Epidemic roseola differs from measles in the shortness of the pre-eruptive stage, the pinker colour and shorter persistence of the rash, the absence of coryza, the very slight fever, and the generally milder course. Previous attacks of measles exert no protective influence.

No treatment of a special kind is called for.

SMALL-POX—VARIOLA.

Small-pox is a specific disease which is chiefly spread directly from proximity to affected persons, or by inoculation with the contents of the pock. There is no reason to doubt that some living organism is the primary cause of variola; but though it has not been identified, recent work points to the probability of its being of a protozoal nature. The poison may also be conveyed to a distance by flies, contaminated clothing, furniture, etc. Small-pox may be met with at any age, and almost exclusively occurs in those who are not protected by efficient vaccination. A second attack is unusual. Besides the eruption, which will be described further on, there are no characteristic anatomical changes.

SYMPTOMS.—The average duration of the stage of *in-*

cubation is twelve days. The *invasion* begins suddenly with well-marked rigor or repeated chills, accompanied by high fever, which rapidly rises to 103° or 104° (*primary fever*). The constitutional disturbance is severe from the outset, and is specially characterised by marked prostration, intense headache, pains in the back and limbs, nausea, and vomiting. With the exception of vomiting, which generally ceases towards the end of the second day, the foregoing symptoms increase up to the appearance of the eruption, but their severity is no safe guide to the subsequent gravity of the case. During this stage prodromal rashes are not uncommon. They are more often of an erythematous type, patchy or diffuse, and sometimes petechial. This rash has usually a limited distribution, and occurs most frequently on the lower part of the abdomen, the upper front of the thighs, and the axillæ. It is probably of septic origin.

The *eruption* generally appears on the third day, and, if later, is rarely if ever confluent. It comes out first on the forehead and wrists, thence extending over the face and arms, and to a less extent over legs and trunk. The number of "pocks" varies much. Each begins as a small isolated congested spot; this enlarges, becomes indurated, and by the third day a *papule* is formed, which feels like a small shot embedded in the skin. Owing to the accumulation of serous fluid in the *rete mucosum*, the horny layer of the epidermis becomes elevated, and by the sixth day a perfect *vesicle* is formed. The vesicle is broad, flattened, and its cavity is subdivided by septa, formed by partially cohering epithelial cells. Owing to the greater exudation of serum and cell proliferation at the periphery of the vesicle, this part is raised above the level of the centre, thus accounting for the central depression of the vesicle known as *umbilication*. A red areola soon surrounds the vesicle, its contents become turbid and finally purulent, the septa break down, and a hemispherical *pustule* is

formed. By these successive stages the period of *maturat-ion* is reached about the ninth day. The eruption is not confined to the skin, but also occurs on the palate, cheeks, fauces, larynx, and sometimes the conjunctiva. The various stages of the eruption in these parts are more rapid and obscure, owing to the softness and moisture of the mucous surface, and ulceration may follow.

In all but the worst cases *the primary fever falls, and the invasion symptoms subside on the appearance of the eruption*. In mild cases the remission is complete. But as the eruption matures, other symptoms appear, the intensity of which is directly proportionate to the number of the pocks. The temperature rises again, representing a suppurative fever akin to septicæmia (*secondary fever*). In “discrete” cases, it lasts from two to four days, and is attended with malaise, headache, sleeplessness, and perhaps slight delirium. In “confluent” cases the remission is less complete and the secondary fever begins sooner, rises higher, and lasts longer; while the systemic disturbance is proportionately more severe. Delirium of an active and violent type is common, merging into collapse. The skin becomes very swollen, red and irritable, especially on the face. Any eruption in the throat will cause soreness; and in the larynx—cough and hoarseness of the voice. A peculiar and disagreeable odour is also apt to emanate from the patient.

The pustules meanwhile dry up (*desiccation*) and give place to dark coloured scabs which usually separate some time during the third week. Since the eruptive inflammation generally involves more or less destruction of the true skin, cicatrices or “pits” occur. These are at first of a dull reddish colour, but ultimately become quite white. With the subsidence of the secondary fever and its accompanying symptoms, convalescence rapidly takes place. It is at this time, however—especially in “confluent” cases—that serious complications are apt to arise.

VARIETIES.—Where the pocks are distinct, and not numerous, the disease is termed *discrete*, and where they are numerous and run together—*confluent*. One of the most serious and fatal varieties is that known as *hæmorrhagic* small-pox. It should be distinguished from the hæmorrhages which not infrequently occur in the pustules as they approach maturation, and which are not of any serious import. The first sign is a petechial eruption, which generally appears on the second or third day, in association with larger ecchymoses, especially round the eyes and beneath the conjunctivæ. Hæmorrhages from the mucous surfaces are common, and the patient quickly falls into a state of collapse, and dies in three to six days. It is worthy of note that, in spite of the virulence of this variety of small-pox, the temperature is not high, and the mind usually remains clear to the end.

COMPLICATIONS AND SEQUELÆ.—The most important are *boils* on the skin, *abscesses*, *erysipelas* of the face and head, *conjunctivitis*, *ulceration of the cornea* which sometimes leads to destruction of the eyeball, *bronchitis* and low forms of *pneumonia*; *laryngitis*, sometimes followed by *œdema of the glottis*, or, in other cases, by *perichondritis* with necrosis of the cartilages in the larynx, and *pyæmia*.

The *diagnosis* will be considered hereafter.

The *prognosis* will turn largely in the first instance upon previous vaccination, its efficiency, and the interval which has elapsed since. As to age (in the unvaccinated) the mortality is highest under five and over forty years of age. On the other hand, a mild primary fever and well-marked remission are good signs. Pregnant women usually abort and die. It is especially dangerous in drunkards, and in the confluent forms of the disease. Epidemic is more grave than sporadic small-pox. The hæmorrhagic form is always fatal.

VARIOLOID.—In those who have been vaccinated, the

course of small-pox is so much modified as to need a separate description under the name of *varioid*. Its main features are as follows:—the rash is scanty and ill-developed; the development of the pocks is much more rapid, and often very imperfect; there is either no “secondary fever” at all, or it is very slight; there is seldom any “pitting,” and the prognosis is almost always favourable. In spite of this mild and shortened course, its recognition is yet extremely important; for, during an epidemic, they may and often do largely contribute to the spread of the disease, because they are often so mild that patients are not confined to the house while retaining the power of transmitting the disease in a virulent form.

TREATMENT.—In mild cases but little is required beyond isolation of the patient, rest in bed, cleanliness and good ventilation, a light nutritious and non-stimulating diet, and general attention to the excretions.

In graver cases we meet with important symptomatic indications. The mouth should be kept scrupulously clean. During the stage of invasion there may be great *nervous excitement* and *delirium*, which may be due either to general excitability, to the height of the fever, or to previous alcoholic excesses. In the *first* form chloral, bromide of potash, and hypodermic injections of morphia are of most service; in the *second*—cold spongings or tepid wet-packs, together with the internal administration of salines and digitalis, frequently repeated; and in the *third*—good nutritious food, with occasional subcutaneous injections of morphia. *Coma*, *nervous prostration*, and *low forms of delirium* require a liberal allowance of stimulants and good food. In such cases I can also speak favourably of tepid sheet-packs, repeated from time to time.

As regards the *eruption*, protection of the surface from air and light by inunctions of different kinds, and keeping

the room darkened, are said to prevent "pitting." It is most important that all crusts should be removed from the face as early as possible, by frequent light meal poultices. A preliminary smearing of the parts with equal parts of oil of eucalyptus and olive oil is recommended. The same object may be attained for other parts by a daily warm bath, in which borax is dissolved. The local application of cold compresses, wrung out in two to three per cent. carbolic acid solution, best relieves the troublesome irritability of the skin. During *desiccation* baths should be taken, and the linen frequently changed. Complications must be treated as they arise. The quarantine period for those exposed to infection is fourteen days; and patients are infective until every trace of scab and scale disappears.

VACCINIA—COW-POX.

Cow-pox is a specific contagious disease of cattle, which is characterised by a pustular eruption closely resembling that of small-pox, and appears in the cow on the udder and teats. It only occurs in the human being as the result of inoculation. The most recent investigations seem to show that vaccinia is in its nature closely allied to small-pox, and represents that disease as modified by successive transmission through the cow. Be this as it may, there can be no doubt that, inoculated into man, it affords substantial immunity from small-pox, which persists for an uncertain period—for many years as a rule—but in a gradually decreasing degree until the protection finally wears out, or unless steps are taken to re-establish it by re-vaccination.

METHODS OF INOCULATION.—*Source of lymph.*—Hitherto it has been obtained from the human being by direct arm-to-arm inoculation; but this source is now being

largely superseded by lymph obtained direct from the vesicles of true vaccinia in the calf, the supply being maintained by successive inoculation from calf to calf. It may be collected in capillary tubes; or ivory points may be charged by dipping them in the lymph, and afterwards allowing them to dry. By either of these methods the lymph may be preserved, and used as required. If obtained from vaccine vesicles in the human being, it should be taken from mature vesicles on the eighth day after inoculation, and unmixed with blood. *Time for inoculation.*—Vaccination should be carried out at two to three months of age, the child should be healthy at the time, and if unsuccessful it should be repeated. *Mode of introducing the lymph.*—The chief methods are *three* in number:—(a) by simple puncture with a lancet; (b) by tattooing; (c) by a series of scratches. The part usually selected is the skin over the deltoid muscle of the shoulder. Liquid lymph direct from the vaccine vesicle, or from a capillary tube, should be inserted by the lancet; with dried lymph on ivory points, the latter should be introduced into the punctures or well rubbed on the surface previously abraded. Whichever method is selected, the punctures or scratches should only penetrate the cuticle. If the deeper layers of the skin be wounded, hæmorrhage will occur, and inoculation will be uncertain. It is usual to inoculate at least four points on the arm, about three-quarters of an inch apart.

PHENOMENA FOLLOWING VACCINATION.—By the *third* day a little red *papule* appears, which becomes *vesicular* by the *sixth* day. The vesicle is roundish, flattened, and generally umbilicated. It is surrounded by a bright red inflammatory areola, and by the *eighth* day is at its height. The contained lymph is clear and slightly viscid, and it is only at this stage it should be used for inoculation. On the *tenth* day the contents become purulent. Shortly after-

wards, the *pustule* begins to dry up, the inflammation subsides, and by the fourteenth or fifteenth day a hard dark scab has formed, which falls off about a week later, leaving a depressed pitted scar, which is permanent. These changes are associated with some general constitutional disturbance and fever. The glands in the axilla often enlarge.

Re-vaccination is recommended about puberty, in order to confer additional protection from small-pox. In some cases no effect results; in others the phenomena above related recur in the usual order, but the development is generally much more rapid, and the different stages are less marked.

The effect of successful vaccination is to confer absolute and complete protection against small-pox for a certain (but variable) time; and if, by reason of lapsed protection, small-pox is contracted, the disease runs a modified and favourable course.

VARICELLA—CHICKEN-POX.

Chicken-pox is an acute specific fever, running a definite course, and is probably only communicable by direct or indirect contact with the affected person. It rarely occurs twice in the same individual, and is generally confined to children.

SYMPTOMS.—The period of incubation is about a fortnight—sometimes a little longer, rarely shorter. The eruption is often the earliest evidence of the disease; but general malaise, restlessness, headache, and slight fever for twelve to twenty-four hours are of common occurrence, and occasionally vomiting and high fever. A prodromal rash is sometimes met with. The eruption at first consists of reddish papules, which quickly become converted into largish single-chambered blebs or vesicles, filled with clear

fluid. About twenty-four hours later the fluid becomes turbid, and the vesicles are surrounded by slight inflammatory areolæ. In a day or two they rupture and dry up, and small scabs are formed, which come away gradually; but, owing to the superficial nature of the sore, "pitting" seldom occurs, unless healing has been prevented by scratching or picking. Some of the smaller papules may abort, without conversion into vesicles, and then gradually disappear. The eruption varies widely in amount, but (unlike small-pox) it is most abundant on the trunk, next on the face, and least on the limbs. It is also important to note that the vesicles appear in *successive crops*. The general symptoms are slight and unimportant, but occasionally convalescence is protracted.

The *prognosis* is always good in uncomplicated cases.

No special *treatment* is called for.

ERYSIPELAS.

Erysipelas is an acute infective disease of the skin. In previous editions of this book it was described as a distinctive disease; but more recent work strengthens the view according to which erysipelas is but one of several septic manifestations, produced by the pus-forming streptococcus, and owes its special clinical features mainly to the fact of its localisation in the skin. It may not only be complicated by cellulitis or pyæmia, but may transmit septic troubles of various kinds to other patients without erysipelatous features. It comes to this—that pus-forming streptococci in the skin produce what we call erysipelas; if they extend to the subcutaneous tissue, cellulitis; if they circulate in the blood, pyæmia; if they gain entrance to the uterus of a parturient woman, puerperal fever; and so on. A frequent mode of introduction is through some breach of the body surface; but, apart from wounds, it often starts

at the junction of skin and mucous membrane, especially at the nose, eye, lips, and ears. Erysipelas is both infectious and inoculable, and may undoubtedly be indirectly conveyed by fomites. General debility, alcoholism, and insanitary surroundings constitute predisposing factors of great importance. So far from conferring protection against its repetition, erysipelas is very liable to recurrence. The period of *incubation* is variable.

MORBID ANATOMY.—The skin becomes red, and infiltrated with serum and leucocytes, leading to various degrees of swelling. Where inflammation is more intense, *vesicles* and *bullæ* may form, containing clear or sanious fluid; or subcutaneous suppuration of a diffuse kind may occur. The lymphatic glands and vessels of the part become enlarged and tender; and the coats of the vessels may inflame, and lead to thrombosis. Inflammation may extend from the skin into the mucous orifices—*e.g.* the ear, mouth, or nose.

SYMPTOMS.—The *local symptoms* are preceded by a period, lasting from three hours to two or three days, which is marked by chilliness or rigors, headache, digestive disturbance, and rapid rise of temperature; and at the end of this premonitory stage the local signs appear. The skin becomes red, swollen, smooth, shining, painful, and tender at some point, starting from which the process rapidly extends, and generally in some special direction. The line of its advance is well defined by an abrupt red margin, while the redness passes quite gradually into the healthy skin, where the inflammation is subsiding. The amount of œdema and pain depends upon the nature of the cellular tissue; where this is dense and unyielding, the swelling is comparatively slight and the pain very severe, while the reverse is the case where the tissue is lax and abundant, as in the eyelids. The starting point of idiopathic erysipelas is generally on the face at the junction of skin and mucous

membrane, especially the *nose*. Thence, it usually extends upwards over the scalp, and the swelling causes much disfigurement, even to obliteration of the features. In bad cases, vesicles and bullæ may form, and subcutaneous suppuration occasionally takes place. Erysipelas may also affect the throat or larynx, causing pain and difficulty in swallowing, and respiratory trouble.

The inflammation usually attains its height on the second or third day; but when the development is slow, it may be at its maximum in one part, while it is subsiding at another. As the swelling and redness abate, the vesicles and bullæ burst and scab over, free *desquamation* of the cuticle follows, and (when the scalp has been effected) the hair usually gets thin, or falls off.

General symptoms.—The temperature rises rapidly to a high grade, but with well-marked morning remissions. The pulse is frequent, full, and strong. In facial erysipelas there is always much restlessness; delirium is of frequent occurrence, and is sometimes of a wild, maniacal type, especially in alcoholic subjects. The tongue is dry and coated, and the appetite is lost. The bowels are usually costive, but troublesome diarrhœa is sometimes met with. The urine is febrile, and generally contains more or less albumen. In intemperate, feeble, or aged patients the disease is apt to assume a “typhoid” or adynamic type, marked by a dry brown tongue, sordes on the teeth and lips, a very rapid pulse, with low delirium merging into a fatal coma. *Defervescence* usually occurs about the end of the first week, when the temperature rapidly falls; but in some cases inflammation may wander about for several weeks. Relapses are common.

The more important *complications* are diffuse cellulitis, meningitis, broncho-pneumonia, infective endocarditis, empyema, acute intestinal catarrh, and œdema of the glottis.

The *prognosis* in uncomplicated cases is generally favourable ; but in drunkards, old people, and infants it is often fatal.

TREATMENT.—Rest in bed, in a room of equable temperature, not too warm, and well ventilated, but free from draughts. Good nourishing food is required, and stimulants are frequently indicated at an early period. Full doses (m20 to m40) of the tincture of the perchloride of iron, frequently repeated, are thought by some to be of value ; but it is very doubtful whether the advance of the disease can be checked by any internal medication. Locally, the inflamed surface should be dredged over with flour, or starch with oxide of zinc, and covered with cotton wool. Such dressing should be bathed off daily with tepid water, and renewed. A ten per cent. ointment of ichthyol is also a good local application. Shaving the scalp, followed by application of ice to the head, is desirable in bad cases with much delirium. In order to allay excitement and to produce sleep, cold sponging and wet cold packs are useful when the fever runs high ; while, as to medicines, bromide of potash and chloral hydrate are recommended in preference to opium. Specific treatment by subcutaneous injection of anti-streptococcic serum (20 cc. once or twice a day) has been tried, but the results hitherto have been disappointing.

GLANDERS—FARCY.

The above disorders can only be communicated to man by direct or indirect inoculation with the virus of a specific organism (*bacillus mallei*), which attacks the horse and animals of that class. Consequently, they are for the most part confined to grooms and stablemen. Glanders and farcy are merely different phases of one and the same disease, the former term being applied to those cases in

which the nares are the primary seat of trouble, while the latter represents a general bacillary infection of the body. The process is usually acute, but chronic forms are occasionally met with.

MORBID ANATOMY.—The characteristic feature of the disease consists in peculiar nodular formations (*farcy buttons*), which vary in size from that of a pin's head to that of a hazel-nut, affecting the skin, mucous membranes, or lungs. They consist of cells, which rapidly soften and break down, forming ulcers when superficial, and abscesses when more deeply situated. The lymphatic glands are often enlarged.

SYMPTOMS.—After an incubation of variable length, the disease sets in with rigors, fever, severe pains in the limbs, and great debility. Before very long a peculiar eruption appears on the skin, which is accompanied with fœtid sweats. In some cases this is preceded by a copious and offensive discharge from the nose. The eruption consists at first of red spots, which soon become indurated and nodular. A pustule then forms, bursts, and leaves an irregular sloughy ulcer with a livid margin. The disease spreads by the lymphatics, and extends to the glands, and internal organs, especially the lungs, where it gives rise to miliary abscesses, or larger pneumonic areas which rapidly break down. There is often more or less erysipelatous redness of the nose, eyelids, and surrounding parts. Meanwhile, the patient becomes progressively weaker and more prostrate, and finally delirium and coma supervene, death usually taking place before the twentieth day. In chronic cases the disease may drag on for many weeks. Acute cases are nearly always fatal.

TREATMENT.—No specific is known. Stimulating, nutritious, and tonic plans of treatment should be carried out. When accessible, all abscesses should be opened, and nodules removed by surgical means.

GENERAL DIAGNOSIS OF ERUPTIVE FEVERS.

Cutaneous eruptions constitute one of the most important diagnostic features of the majority of the acute specific fevers; and, by their differences as to date of

	MEASLES.	SCARLET FEVER.	ERYSIPELAS.	GERMAN MEASLES.
INITIAL SYMPTOMS.	Those of common catarrh (swelling of face and eyelids, coryza, photophobia, lachrymation and cough). Moderate fever. Koplik's spots.	High fever, pungent heat of skin, and great frequency of pulse, which set in suddenly and develop rapidly. Initial vomiting is common.	Fever marked in proportion to the eruptive inflammation.	Moderate fever, slight constitutional disturbance. No coryza. Frequently slight enlargement of cervical glands.
RASH.	Appears on 4th day. Begins on forehead. Best marked on the exposed parts. Dusky red crescentic patches.	Appears on 2nd day. Begins on the neck. Best marked on the covered parts. Bright red points merging into large areas of redness.	Appears in a few hours to 2 or 3 days, and begins generally on the nose or eyelids, and extends thence in one definite direction, generally upwards over the head.	Appears on 1st day. Begins on face. Spots do not merge into large areas of uniform redness. Considerable irritation of skin.
TONGUE.	Furred.	Red, with prominent papillæ.	Furred, tendency to get dry and brown.	Not characteristic.
THROAT.	Slightly sore.	Soreness generally marked; diffused redness of the fauces and soft palate.	Unaffected, as a rule.	Slightly sore. No ulceration.
DESQUAMATION.	Slight and branny.	Free and persistent.	Free.	Slight and unimportant.

onset, site of first appearance, and general characters, afford a ready and useful means of distinguishing one fever from another.

If the rash appears within the first four days from the onset of the fever, it is either scarlet fever, measles, epidemic

roseola, erysipelas, small-pox, chicken-pox, or glanders. The onset of each is usually well marked, and may be recognised, not only by increased heat of the body, but also by the occurrence of rigors, vomiting, or convulsions. *If the rash consists of patchy, or more or less diffused redness*, which disappears readily on pressure, and is not attended with any infiltration of the skin, it is either scarlet fever, measles, epidemic roseola, or erysipelas; and such specific red rashes may be distinguished from simple roseola, by the very sudden appearance and fugitive character of the latter, the slight febrile disturbance, the absence of sore-throat, the mild constitutional disturbance, and non-infective origin.

If the eruption be papular in the first instance it is either small-pox, chicken-pox, or glanders.

	SMALL-POX.	CHICKEN-POX.
INCUBATION.	Constant, lasting 12 days.	Seldom under 14 days.
ERUPTION.	Appears on 3rd day. Begins and is most abundant on the face and limbs. Comes out in one crop. Frequently present on mucous membrane. Begins as well-marked shotty papule; afterwards a vesicle which is flattened, umbilicated, multilocular; ultimately a pustule which is matured by the 9th day.	Appears within 24 hours. Begins and is most abundant on the body. Comes out in successive crops. <i>Very rarely</i> present on mucous membranes. Papules imperfect, and become vesicular in the course of a few hours. Vesicles hemispherical, one-chambered, not umbilicated, and do not become pustular.
INITIAL SYMPTOMS.	Pain in the back, vomiting, and high fever.	Very slight indeed, and often not observed at all.
DURATION.	Three to five weeks.	Average 7 to 10 days.

Glanders is distinguished from the other two by the history of the case (as a rule it is confined to grooms or stablemen); while the rash differs in appearance and is followed by characteristic rapid ulceration.

The only other eruptive fevers are typhus and typhoid, which may be thus contrasted:—

TYPHUS FEVER.	TYPHOID FEVER.
May occur at any age. Invasion more or less abrupt. Face dusky; expression heavy; and eyes injected and suffused. No special abdominal symptoms, or diarrhœa. Prostration and low nervous symptoms appear early. Special eruption which appears on the 4th or 5th day; first on the back of wrist, and epigastrium; comes out in one crop; and does not disappear on pressure. Special course of temperature. (Refer.) Duration shorter than that of typhoid.	Rare after middle age. Invasion gradual. Face pale, with a circumscribed flush on each cheek. Typical abdominal symptoms, and diarrhœa as a rule. Low nervous symptoms, if present, appear much later. Special eruption which appears from 7th to 12th day; first on the abdomen or back; comes out in successive crops, and disappears on pressure. Special course of temperature. (Refer.) Duration longer than that of typhus.

YELLOW FEVER.

This is a specific fever, characterised by toxæmic jaundice. It does not appear to be directly infectious from one person to another; and doubt has been recently thrown upon its conveyance by fomites, at any rate apart from favouring conditions, such as high temperature, humidity, and insanitation. Although quite distinct from malarial disease, it is probably inoculable by mosquito-bites. No specific organism has yet been identified with the disease. Yellow fever is endemic in some areas, especially the West Indies, and certain parts of the Brazilian coast. Epidemic outbreaks in certain tropical littoral districts are of frequent occurrence; but isolated epidemics, often severe, do sometimes occur in relatively temperate climates. Susceptibility is much greater in white than in coloured races. One attack is almost invariably protective against another.

MORBID ANATOMY.—There are no distinctive lesions. The body-surface has a yellow colour, with hæmorrhagic spots and patches, which are most marked on the dependent parts. The stomach and intestines contain black

disintegrated blood and mucus ; their mucous lining is softened and injected ; and degenerative changes are often met with in the liver, heart, muscle, and other parts.

SYMPTOMS.—With or without warning the invasion is sudden, and marked generally by rigors. The temperature rapidly rises to a variable height (101° to 105°), which is maintained *without remissions* for three or four days. There are the usual febrile symptoms, beyond which the most marked features are troublesome vomiting, tenderness of the epigastrium, intense frontal headache, redness and irritability of the eyes, traces of jaundice, with pains in the back and limbs. The urine generally contains albumen.

On the third or fourth day, the temperature falls and the symptoms subside, with remarkable slowing of the pulse down to forty or less ; but the improvement is of short duration, except in very mild cases when the disease may pass off altogether. Secondary febrile reaction soon takes place, epigastric tenderness is increased, the vomit contains blood (“black vomit”) and the stools are black from the same cause. Marked jaundice now supervenes, petechiæ may be observed, hæmorrhages from mucous surfaces are not uncommon, and the general condition is one of great prostration. The further course varies. In severe cases, the patient speedily passes into a state of collapse, with scanty and highly albuminous urine, more or less delirium merging into coma, and death somewhere between the fifth and tenth day. In favourable cases, however, improvement sets in, about the tenth day as a rule (sometimes with profuse sweating), and the patient slowly recovers. Relapses are not uncommon, and are often fatal.

DIAGNOSIS.—A bilious form of remittent fever may closely resemble yellow fever, but the former is not contagious, does not confer protection against a second attack, there are frequent remissions of temperature with sweating

at short intervals from the beginning of the disease, there is enlargement of the spleen, hæmorrhages are not so severe and persistent, and the disease is controlled by quinine.

The *prognosis* varies greatly in different epidemics. Death sometimes occurs in the early febrile stage from sudden collapse. In the second stage the gravity is proportionate to the prominence and persistence of hæmorrhagic and nervous symptoms.

TREATMENT.—The food should be light, liquid, and nutritious, and alcoholic stimulants are of great value. If these cannot be retained on the stomach they should be given *per rectum*. Medicinally, Sternberg's mixture is recommended, consisting of sodium bicarbonate, gr. 150, and hydrargyrum perchloride, gr. 1, dissolved in a quart of water. Three tablespoonfuls should be given every hour. A single dose of castor oil or calomel is advised at the outset, but afterwards only enemata should be used. Vomiting is best relieved by ice-pills, and sinapisms to the epigastrium; fever, by frequent sponging; nervous symptoms, by tepid sheetpacks; and collapse, by subcutaneous saline injections. Opium should be avoided. Great care in feeding should be observed during the period of remission; and return to solid food during convalescence should be very gradual.

CEREBRO-SPINAL FEVER.

Cerebro-spinal fever is an acute specific disease, which is characterised by inflammation of the membranes of the brain and spinal cord. It may occur in an *epidemic*, or a *sporadic* form. It is not directly contagious. It is believed to be due to a special organism (*Diplococcus intracellularis*), but very little is known as to the conditions which determine its appearance or propagation. Young persons are most liable to be attacked; and the posterior basic meningitis of infants is probably of this nature.

Its *morbid anatomy* consists of inflammation of the pia mater of the brain (especially at the base) and cord, attended with a purulent or semi-purulent exudation into the sub-arachnoid tissue, and an excess of somewhat turbid fluid in the ventricles. The substance of the brain and cord is congested.

SYMPTOMS.—The disease usually *commences very abruptly* with a violent chill, intense headache, and persistent vomiting. Stiffness and retraction of the neck, pain down the spine and radiating to the limbs (much increased on movement), and general cutaneous hyperæsthesia are also early and characteristic symptoms. The head is usually retracted, and there may be more or less tonic contraction of the extensor muscles of the neck and spine. There is almost always a certain amount of delirium, often of a violent type, which sometimes alternates with insensibility. In very mild cases, however, the patient may be merely dull, drowsy, and restless. The face is pale and the expression is anxious. The pyrexia, though variable, is usually moderate in amount, and runs a most irregular course.

The disease now rapidly develops to its full intensity. About the second to the fifth day various cutaneous eruptions may appear. Of these, *herpes* on the face and lips is the most common; but petechiæ, and small patches of erythema, or roseola, are not infrequently met with. There is, as a rule, more or less intolerance of light and sound; and ptosis, strabismus, or irregularity of the pupils may be sometimes observed. Tinnitus and partial deafness are also not uncommon. The pulse is feeble, and liable to great variation in frequency; the tongue is brown and dry; the appetite fails, and the bowels are constipated. When the thigh is sharply flexed on the trunk, there is great resistance to the extension of the leg at the knee (Kernig's sign).

In fatal cases the symptoms of irritation subside, and are replaced by those of general depression; death taking place in a few days from coma. In favourable cases the signs of depression are either absent, or comparatively slight. The symptoms gradually subside, and convalescence occurs in two to four weeks; but it may be protracted for a long time, with headache, pain in the back, or disturbance of the sight or hearing.

The most important *sequelæ* are deafness, derangement of vision, chronic hydrocephalus, and chronic meningitis. The mortality varies within such wide limits as from twenty to eighty per cent.

TREATMENT.—The most important indications in the acute stage are—rest in bed, cold applications to the head, local abstraction of blood by leeches placed behind the ear, and the free and early hypodermic injection of morphia. In the later stages, iodide of potassium combined with tonics appear to be of some service.

INFLUENZA.

Influenza is an acute specific disease occurring for the most part in epidemic form, travelling with great rapidity, and affecting large numbers in an incredibly short period. There is little doubt but that influenza is due to minute highly infective bacilli, discovered by Pfeiffer, which can almost invariably be found in the bronchial and nasal secretions, but are absent from the blood. The disease is especially fatal among the aged, and those whose vitality is depressed by alcoholism, malnutrition, insanitation, or previous disease. There are no characteristic anatomical changes. It confers no protection against a second attack.

SYMPTOMS.—Owing to the great variety of its manifestations, a short and clear description of influenza is a very difficult task. In a typical uncomplicated case, after a

short incubation period of uncertain duration the disease begins abruptly with chilliness or actual rigor, severe headache, aching pains in the back and limbs, followed by high fever and great prostration. On the second or third day, as a rule, the temperature begins to fall, and in favourable mild cases there may be no other symptoms, and strength is slowly recovered. In some cases, the disease assumes a catarrhal type, with coryza, suffusion of the conjunctivæ, slight sore throat, and bronchial catarrh. Such symptoms may gradually pass off, and end in recovery, but very often (perhaps as the result of the patient leaving his bed too soon) broncho-pneumonia supervenes, with rather peculiar characters. There is irregular patchy impairment of resonance, but no well-defined dullness; the breath-sounds are weak, or sometimes imperfectly bronchial, accompanied with high-pitched sticky crepitations, most marked over the posterior bases. These physical signs have a curious way of creeping about—now in one place, then in another; in fatal cases, gradually extending in area, but not altering much in character, with increasing distress of breathing, feebleness of pulse, prostration, and often with delirium, until the end arrives, after a very variable duration. In other cases, again, the incidence of the disease falls mainly upon the gastro-intestinal tract, giving rise to complete anorexia, abdominal pain, vomiting, diarrhœa, and occasionally jaundice. More rarely there is pronounced nervous disturbance from the outset, with early prostration and delirium, terminating in coma and death. Herpes and various erythematous eruptions are not uncommon.

Complications and sequelæ are especially numerous. Among the more important are the following: prolonged weakness; intermittent attacks of pyrexia, preceded by shivering and followed by profuse sweats; broncho-pneumonia; pleurisy and empyema; functional depression

of the heart ; troublesome neuralgia ; nervous depression ; and mental alienation.

TREATMENT.—No lowering measures are admissible. The diet should be nutritious and liberal, regulated according to the digestive capacity. Alcohol is necessary in a large proportion of cases, especially among the aged, feeble, and intemperate. Medicinally, ammoniated tincture of quinine, the salicyl compounds, phenacetin, or phenazone often give great relief. Bromide of ammonium, in combination with tincture of henbane, is useful for the relief of restlessness and insomnia ; but sometimes, especially among the aged, an opiate at bedtime for the first few nights answers better. Complications demand the same treatment as when they occur under other conditions. Lastly, during convalescence, good nourishment, change of air, and medicinal tonics are indicated.

DIPHTHERIA.

Diphtheria is an acute specific disease, epidemic and contagious, and is usually characterised by a membranous exudation on the mucous membrane of the throat or adjacent parts. Occasionally, a wound on the surface of the body may be the seat of diphtheritic infection. The disease is not only conveyed directly by contact with affected patients, but indirectly by infected clothing and other external objects. It is primarily caused by a specific bacillus (*Klebs-Loeffler*), which is mainly found at the seat of infection, and yields toxins, to the absorption of which the phenomena of the disease are due. The bacilli vary in length, are straight or slightly bent, non-motile, often with rather swollen ends, and do not form spores. They are sometimes found in the throats of healthy persons, who are insusceptible ; and are apt to persist for weeks or months in the throat after the disease has passed off.

Associated with these specific bacilli are pus-forming streptococci and staphylococci, which are very probable factors in some of the inflammatory complications. The disease is most common among the young, but no age is exempt.

MORBID ANATOMY.—The inflammation is more often limited to the throat, affecting the tonsils, soft palate, or pharynx; but it may spread to the nose, larynx, and air-passages. On the other hand, it sometimes begins in, and may be confined to, the larynx or nose; while still more rarely, it attacks the conjunctivæ, the external auditory meatus, or the skin. The mucous membrane is at first red, swollen, and coated with viscid mucus. The inflammation extends, and at one or more points a fibrinous exudation appears, patches are formed, and ultimately the entire surface of the throat may be thus covered with what is called “false membrane.” The latter is at first quite thin, and easily removable from the surface on which it lies. Soon, however, it increases in thickness, becomes opaque, yellowish in colour, and more or less firmly adherent to the subjacent mucous membrane, the superficial layers of which it infiltrates. It consists of fibrin, in which epithelial and red blood cells, leucocytes, and clumps of bacilli are embedded. Later on, the whole exudation undergoes coagulation-necrosis, and comes away, leaving a sloughy surface which in favourable cases soon heals. The profound effects of diphtheria toxins in the production of degenerative changes in the heart-muscle, kidneys, and nervous system should be borne in mind.

SYMPTOMS.—Diphtheria may begin abruptly with a severe rigor and vomiting; but much more frequently the onset is gradual and marked with chilliness, depression, loss of appetite, and general malaise, and is soon followed by more or less pyrexia, soreness of the throat, stiffness of the neck, painful or difficult deglutition, and slight swelling of

the glands at the angle of the jaw. The throat at this stage is red and swollen, with one or more small greyish-white or yellowish spots on the tonsil, soft palate, or back of pharynx. By about the third day these initial spots and patches have extended and thickened so as to form a more or less continuous "false membrane" over the fauces and soft palate. Meanwhile the throat becomes swelled, and the glands of the neck more enlarged and tender, so that swallowing is both painful and difficult. The pyrexia, though sometimes high, is usually quite moderate; the pulse is rapid and feeble, the appetite is lost, and the general quietness and depression of the patient (especially in children) contrasts strongly with the restlessness and irritability of scarlatina anginosa. The urine is generally albuminous from the outset. More often the exudation does not extend beyond the area above mentioned; and in favourable cases (which are more frequent than sometimes stated) a change for the better begins on the fourth or fifth day, the membrane separates, and a few days later convalescence begins.

In a certain proportion of cases the course is much more severe than this—either by the extension of the disease into the pharynx, nose, larynx, or air-passages, or by special severity of the constitutional disturbance. If the nose is affected, there is more or less obstruction of the nostrils with a foetid discoloured discharge. The Eustachian tubes and ear may be implicated. A still more serious extension is into the larynx producing signs and symptoms which will be discussed under the head of "Croup" in a later chapter. From this point a further extension into the bronchi and lungs is of frequent occurrence, giving rise to a specially fatal bronchitis or broncho-pneumonia. Constitutional disturbance is also occasionally so severe that the patient is overwhelmed in the first few days, before the local changes have fully developed. In other cases, which are

less rare, though the local condition may have improved, the patient may suddenly die from syncope. Delirium is exceptional.

In the foregoing account it has been taken for granted that a "false membrane," as described, is characteristic of the disease. While this is true of by far the majority of cases, it must be remembered that the local inflammation is sometimes comparatively mild, and that consequently the exudation is much less marked—so much so that, apart from bacteriological examination of the secretions, or the occurrence of other obvious cases at the same time, their true nature might easily escape recognition.

Liability to fatal heart failure is one of the most serious risks in diphtheria, and is not confined exclusively to severe cases. It results from degenerative changes in the cardiac muscle and the vagus nerve. It may occur early or late in the course of the disease. The early form is most common in cases which are severe from the outset, and probably depends mainly upon progressive cardiac degeneration. The action of the heart becomes steadily weaker, with recurrent attacks of syncope, any one of which may be fatal. Cardiac failure may also occur with startling suddenness in the third or fourth week from the onset of the disease—often in cases which, so far, appear to be doing well. An unexplained increase in the frequency of the pulse, vomiting, pallor, and sometimes precordial pain are very suggestive of such a possible danger, but fatal syncope may occur without any warning of this kind.

Peculiar nervous sequelæ of a paralytic nature also not infrequently occur (*even after the mildest attacks*) during the second or third week of apparent convalescence. The more common forms are paralysis of the soft palate and pharynx, causing a nasal tone of voice, regurgitation through the nose on drinking, and difficulty in swallowing; of the ocular

muscles, causing loss of accommodation, or squint ; of the limbs (especially the legs), causing more or less loss of power ; or of laryngeal muscles, causing aphonia. There is frequently also some impairment of sensation. Recovery is the rule, but it may be slow ; and death sometimes occurs from implication of the respiratory muscles. These paralyses are probably due to degenerative changes in cells of the anterior cornua of the cord, of toxic origin.

DIAGNOSIS.—In well-marked cases the diagnosis is easy ; and, in all cases of doubt, it has now become the rule to abide by the results of bacteriological examination, and meanwhile to take all necessary precautions.

TREATMENT.—Reference has already been made (p. 40) to the method of “serum treatment” which is now employed for diphtheria with such good results. The antitoxic serum—as it is called—is procured from horses, previously immunised by inoculation with diphtheria toxin. The strength of this serum is expressed in “units,” each of which represents the amount which, when mixed with 100 times the fatal dose of toxin, protects a guinea-pig of 250 grammes weight from death within four days (Ehrlich). It is administered by subcutaneous injection in a minimal dose of 1,000 units, but 2,000 units is only an average dose, and in severe cases, 4,000–6,000 units are needed. This dose should be repeated, as a rule, in twenty-four hours ; and sometimes a third dose is desirable. The sooner the treatment is begun after the onset of the disease the better are the results. If given under cleanly conditions, no serious effects need be feared ; but erythematous rashes, joint pains, and pyrexia are not uncommon. In favourable cases the condition of the throat improves and the membrane begins to disappear within twenty-four hours, while the general symptoms begin to abate.

The serum treatment should be supplemented by general and local measures. Absolute rest in bed, pure air, alcohol

in some form, and good nourishment are urgently needed from the outset. Beef tea (strong) and milk should be given at short intervals; and if from any cause food cannot be taken by the mouth, it should be given by rectum or nasal tube. Alcohol in moderate amount is desirable in all but the mildest cases; and in severe cases should be given more freely. As to medicine, strychnia is useful, and may be combined with the tincture of perchloride of iron, or (as recently recommended) a 25 per cent. solution of formic acid in 5-15 minim doses. As to local treatment, strong caustics are both unnecessary and harmful. Since the introduction of serum treatment, throat applications are not of the same importance as formerly; but it is desirable to keep the mouth, throat, and nasal passages as clean as possible, and for this purpose simple antiseptic washes, such as solution of boric acid (2 to 3 per cent.), mercury perchloride (1 in 3,000), or peroxide of hydrogen (5 to 10 volume solution), may be used with a syringe, douche, or spray apparatus. Great comfort is also derived from sucking ice constantly and freely. As affording some protection against heart failure, alcohol and strychnia are of most service, coupled with absolute rest. At the time of a syncopal attack little more can be done; but the foot of the bed should be raised, and hot applications over the heart are advised. With obstinate vomiting, the patient should be fed per rectum. Extreme restlessness and sleeplessness may be combated with an opiate combined with atropine or belladonna. In no case should the patient be allowed to sit up (even in bed) under three weeks. During convalescence, quinine, iron, and change of air are required. The nervous sequelæ are best treated by strychnia, galvanism, and the application of blisters alongside the spinal column.

For purposes of *prophylaxis*, nothing is better than daily spraying of the throats of those exposed to infection with

a saturated solution of boracic acid. The injection of antitoxin (1,000 units) may be employed with advantage in some persons, such as those in immediate attendance on the patient.

MUMPS—EPIDEMIC PAROTITIS.

Mumps is a specific infectious disease, especially prevalent amongst young people. It is probably caused by some specific organism, of which we have, as yet, no definite knowledge. It is highly infective at close quarters; and though it may be carried indirectly through a third person, infectivity in this way is relatively slight.

It is characterised by inflammation of the parotid gland, but suppuration very rarely occurs. As a rule, swelling of the parotid and adjacent tissues, between the mastoid process and lower jaw on one side, is the first sign of the disease; but occasionally this is preceded by general malaise and perhaps some earache for a day or two. In any case the onset is usually marked by a sharp rise in temperature. The swelling increases, and extends downwards, becoming hot and tender, thus rendering all movements of the jaw painful and difficult. In two or three days, the opposite side becomes involved in a similar way. The submaxillary gland is sometimes attacked, and the cervical glands are often enlarged. The swelling generally soon begins to subside, the temperature gradually falls, and by the end of seven to ten days the patient is convalescent. Not unfrequently, as the inflammation is subsiding in the parotid, it suddenly attacks the testicle (usually only on one side), giving rise to an orchitis which is attended with a recrudescence of fever and considerable constitutional disturbance. It usually lasts three or four days, and is sometimes followed by wasting of the organ. The author has seen two cases in which the mammary

gland was attacked in like manner during parotitis. Ovaritis may also occur, but is rare. Under similar circumstances acute pancreatitis has been observed in some recent epidemics, characterised by vomiting, pain and tenderness in the epigastrium, and constipation. It is worth remembering that deafness sometimes follows an attack of mumps.

The *prognosis* of the disease is almost uniformly favourable. In adults, however, it is apt to be more severe than in children, and convalescence more protracted.

The *treatment* is exceedingly simple. Rest in bed for at least a week, a mild aperient, together with hot fomentations to the affected part include all that is generally required. Twenty-four days quarantine is recommended, dating from exposure to infection.

WHOOPIING-COUGH.

Whooping-cough or pertussis is a specific infectious and often epidemic disease. The contagion on which it depends has not been identified with certainty, but it appears chiefly to fasten upon the mucous lining of the throat and upper air-passages. It is undoubtedly present in the sputa, by which the disease may be conveyed to others; but it may also be carried by clothing, articles of furniture, and the like. A quarantine period of twenty-one days from exposure to infection is recommended; but there is little danger of infectivity later than six weeks from the date of onset, provided the patient and clothes have been disinfected. A recent attack of measles—and, in fact, any catarrh of the respiratory tract—seems to favour the occurrence of whooping-cough, when the latter is prevalent. It is essentially a disease of childhood, but adults are not exempt. A second attack is rare. The anatomical appearances have no characteristic features.

SYMPTOMS.—After an incubation of seven to fourteen days the disease sets in, with a course which may be divided into three stages. The *catarrhal* stage develops gradually with the signs and symptoms of an ordinary “cold,” with slight fever, cough, and expectoration. The cough is notably more troublesome at night. These symptoms continue for about a week or longer, steadily becoming worse. As the *paroxysmal* stage begins, the catarrhal secretion often diminishes, but the cough gets worse, lasts longer, and assumes a characteristic paroxysmal form. Each fit consists of a series of short, sharp, rapidly successive coughs, followed by a prolonged crowing inspiration or “whoop.” The paroxysm may be repeated once or twice, until some viscid mucus is expelled, or actual vomiting occurs, which brings it to an end. The attack is followed in infants by great exhaustion, but in older children this is not so marked. The nervous agitation and distress which immediately precede the paroxysm are sometimes very remarkable, and characteristic. Owing to the obstructed circulation during the paroxysm, the face is dusky, the veins become swollen, the eyes suffused, and suffocation seems imminent. Hæmorrhages from the nose, mouth, ears, or beneath the conjunctivæ are apt to occur. The *frænum linguæ* may be ulcerated as the result of forcible and repeated protrusion of the tongue over the lower incisor teeth in the act of coughing; and hernia may be sometimes induced. The daily number of paroxysms varies widely, but they are usually worse and more frequent at night.

After the lapse of two to three weeks the stage of *decline* gradually sets in, the coughing fits become fewer and less severe, the bronchial secretions become more abundant, easy, and muco-purulent, until convalescence is re-established. The physical signs—in the absence of pulmonary complications—are unimportant.

Simple pertussis, though often protracted, is rarely fatal ; but in the presence of complications (which are common in young children) the prognosis may be very serious. The complications fall into three main groups. The first and most common are characterised by *gastro-intestinal disturbance* such as frequently repeated vomiting and diarrhœa. *Nervous complications* are scarcely less common, especially convulsions. Though not necessarily serious, they often indicate the occurrence of such grave conditions as intra-cranial hæmorrhage, thrombosis of cerebral sinuses, broncho-pneumonia, or extensive collapse of lung. The last two represent the most important of the *pulmonary complications*, and materially increase the risk of a fatal termination. Adenoid overgrowth in the naso-pharynx not infrequently date from an attack of whooping-cough.

DIAGNOSIS.—The peculiar paroxysms are quite characteristic.

TREATMENT.—As soon as the true nature of the disease is suspected, the child should be isolated in a warm airy room. Thorough ventilation is essential, and it is wise to have separate rooms for day and night accommodation when practicable. Impregnation of the air with the vapour of carbolic acid is helpful. For this purpose, a five per cent. solution of crude carbolic acid should be volatilised in a bronchitis-kettle placed over a lamp near the bed of the patient. The diet should be light and nutritious, but care should be taken to avoid overloading the stomach, especially when vomiting is troublesome. During the catarrhal stage, some simple saline with vin. ipecacuanhæ and paregoric, warm baths, and counter-irritation to the chest are advisable. In the paroxysmal stage, the best medicine is carbolic acid (℥ $\frac{1}{2}$ -j. with a little mucilage, glycerine, and aromatic water, every two or three hours). In many cases, especially where there is much nervous excitability and

sleeplessness, bromide of potassium with belladonna are useful, to which a few grains of chloral hydrate or phenazone may often be added with advantage. When vomiting is so frequent as to interfere with proper nourishment, occasional spraying of the throat with a two per cent. solution of cocaine hydrochloride may give great relief. When this is impracticable, it may be given by the mouth (one sixth of a grain twice a day, for a child of a year old). If the weather is sufficiently favourable, and in the absence of complications, the little patient may be allowed out of doors with advantage, in the later stages of the disease.

During the decline, cod-liver oil, tonics, and change of air are very desirable.

EPIDEMIC CHOLERA.

Cholera is an *epidemic* disease, sometimes *endemic*, and probably dependent on a *specific organism*, in the form of small curved rods (comma bacilli), discovered by Koch in 1884, who found them in the intestinal contents and discharges. The exact conditions under which they exist and grow outside the body have not been determined. Koch's views have not yet received general acceptance, and it may be that other conditions are necessary to determine the occurrence of the disease. But at any rate, the stools are the main, if not the only, source of infection, which is usually conveyed through the medium of contaminated drinking-water. It is probably not conveyed through the air. Among reputed favouring conditions, attention is called to high temperature, humidity of the air, insanitation, fatigue, and debility.

The disease affects persons of all ages, and its occurrence does not confer protection against a second attack.

ANATOMICAL CHARACTERS.—When death occurs during the stage of collapse, rigor mortis sets in early, the body is

shrivelled and shrunken, and the dependent parts are congested. The blood is thicker and darker than usual; the pulmonary artery, right side of the heart, and systemic veins are generally gorged with blood, while the left side of the heart is nearly empty. The spleen is small. The intestinal mucous membrane has a sodden appearance, and its follicles are enlarged and prominent. The contents of the bowels resemble the discharges during life, added to which there is abundance of shed epithelium (probably a post-mortem change). The large bowel is usually contracted.

If death occurs during reaction, the blood is more equally distributed, and secondary inflammations may be met with in the lungs and elsewhere.

SYMPTOMS.—After an *incubation* of uncertain length, there are occasionally premonitory symptoms for a day or so, characterised by malaise, depression, and slight diarrhoea, but the onset is more often sudden.

1. *Evacuation stage.*—Purging (with or without pain) is the first symptom. The stools are watery and profuse; and though at first fæcal, soon become thin, neutral or alkaline, colourless, and almost odourless, with whitish flakes in suspension (*rice-water stools*). The quantity of fluid thus discharged is often enormous. Vomiting comes on a little later. The vomit is at first bile-stained, but afterwards consists of thin watery mucus, and may be very abundant. Painful cramps in the limbs and abdomen and intense thirst are generally observed, culminating in the collapse which is characteristic of the next stage.

2. The "*algide*" stage is marked by profound collapse. The tissues shrink, the features are pinched, the eyeballs become sunken and the cheeks hollow. The surface is more or less livid, cold, and clammy. The surface temperature falls to 94° or 95° or lower, while in the rectum it may be 101° or higher. The pulse is small, rapid, and

almost imperceptible; the respirations are rapid and shallow; the expired air is cool; and the voice is faint and whispering. The various secretions of the body are greatly diminished, or totally suppressed. The patient throws his arms about, becomes restless and wakeful, and is singularly apathetic. He complains of intense thirst and heat at the epigastrium. Finally, the vomiting and purging cease, or are greatly diminished, and the patient lies helpless as a corpse. The mind is clear at first, but in fatal cases stupor sets in, followed by coma.

The *duration* of this stage is from two to twenty-four hours, and the symptoms also vary much in intensity. In severe cases they rapidly develop, and death occurs from coma, most often between the tenth and fourteenth hours. In more favourable cases reaction may take place.

3. The *stage of reaction* sets in gradually with general improvement, the pulse becomes stronger, the respiration slower, and the skin loses its livid and shrunken aspect. The thirst, diarrhœa, and restlessness subside; the secretion of urine returns, and the stools are once more coloured with bile. The body temperature is more equally distributed—that of the surface rises, while that of the internal parts falls. The patient may fall asleep, and in mild cases recovery may be re-established in the course of twelve to twenty-four hours. More often, however, the reaction assumes a febrile type, and is more severe and prolonged (“cholera-typhoid”).

It thus appears that, in cholera, we have to do with an extremely acute intestinal catarrh, which is attributable to the intense irritation set up by the comma bacillus. So far as the actual symptoms are concerned, there is nothing to distinguish them from those of equally severe acute intestinal catarrh set up by other causes. No doubt the toxins of the cholera organism directly contribute to the

collapse, and to some of the complications ; and, in rare cases, may determine a fatal issue before diarrhœa has begun. It is impossible, however, to differentiate such effects clinically ; and hence the difficulty of *diagnosis* apart from other evidence of a climatic, epidemic, or bacterial nature. The *complications* also of cholera, such as suppression of urine, persistent diarrhœa, low forms of pneumonia, and cardiac thrombosis, are not characteristic, and are related mainly to the serious diminution of the body-fluids, and the extreme prostration caused by the disease. Pregnant women nearly always abort, if attacked by cholera.

The *prognosis* is necessarily very grave, and the mortality is on the average about fifty per cent.

TREATMENT.—It is impossible to over-estimate the importance of *preventive treatment*, by avoidance of infected localities, by careful attention to purity of drinking-water, strict sanitation, domestic cleanliness, and immediate disinfection of all evacuations and contaminated articles. All diarrhœas during an epidemic should receive prompt attention.

No remedy or system of treatment is as yet known for the cure of cholera when once established. At the same time, by attention to certain general principles and symptomatic indications, much may often be done to promote the comfort of the patient, and to assist in his recovery. He should rest in bed in a clean, warm, and well-ventilated room, and have ice to suck. If the vomiting is severe, it is useless to give any nourishment by the mouth ; but a little chicken or veal broth, or diluted meat-juice, may be tried if it can be retained. A mustard compress should be applied to the abdomen ; and frictions and hot-bottles should be used to maintain warmth. Subcutaneous injection of morphia, pil. plumbi cum opio, or chlorodyne may be given at the outset, to relieve pain and restlessness, and

repeated as required, but in the stage of collapse it is safer to avoid opiates of any kind. In severe collapse, copious injections of saline fluid *per rectum*, or subcutaneously, are recommended.

When *reaction* sets in the diet should be very bland and light, and only increased and improved with great care. Stimulants are not necessarily required, but should the "typhoid state" supervene, they are beneficial. Water may be freely allowed, to which the addition of salt and carbonate of soda is recommended. Complications and sequelæ must be treated as they arise. Tonics and preparations of iron are often serviceable during convalescence.

DYSENTERY.

In the present state of knowledge, dysentery must be regarded as a clinical term, characterised, as a rule, by frequent discharges of mucus and blood from the bowels, accompanied with tenesmus (straining) and abdominal pain. Probably several distinct affections are included under this term. In many cases the disease is undoubtedly set up by a specific pathogenic organism; and, though the bacteriology of dysentery is still incomplete, we are justified in recognising at least two specific forms of the disease—one of bacillary, and another of amœbic origin. But non-specific cases, due to intestinal irritants of various kinds, are of common occurrence. Dysentery is often *endemic* in malarial regions; and also occurs in an *epidemic* form both in tropical and temperate climates, especially in connection with military encampments, under insanitary conditions and overcrowding. In other cases, it is *sporadic*, and may be traceable to decomposing food, or tainted drinking-water. Broadly speaking, endemic and epidemic dysentery are of a specific nature, the former being due to a special

B. dysenteriae (Shiga), the latter to an *amæba*; while sporadic cases—at any rate in this country—are of a non-specific nature. There is little doubt that the disease is infectious through the excreta, and that the contagium may be conveyed by contamination of food or drinking-water. It occurs at all ages; and no race is exempt.

MORBID ANATOMY.—The essential feature consists in an inflammation of the mucous membrane of the colon, especially towards its lower end. The small intestine is sometimes implicated. The local appearance chiefly depends on the severity of the inflammatory process. In mild cases there are patches of catarrh, characterised as usual by congestion and swelling of the mucous membrane, mucous exudation, and cellular proliferation. The solitary follicles are unduly large and prominent. Superficial erosions or follicular ulcers may occur here and there, the peritoneal aspect of which may exhibit signs of slight inflammation. In severe cases the inflammation is of the diphtheritic type, and is characterised by patches of fibrinous exudation on the surface and into the substance of the mucous membrane, followed by necrosis and ulceration. As a rule, only the mucous membrane is involved, but the slough sometimes includes the deeper tissues and may thus lead to hæmorrhage or perforation. In favourable cases the ulcers ultimately cicatrise, but the process is liable to be followed by thickening, puckering, and more or less contraction of the intestine. In chronic cases the coats of the lower bowel become persistently thickened and irregular, often exhibiting evidence of ulceration in various stages, attended with a muco-purulent discharge.

Tropical abscesses of the liver, enlargement and softening of the mesenteric glands, bronchitis, pneumonia, etc., are not infrequently met with in association with dysentery.

SYMPTOMS.—The symptoms of acute dysentery—like the morbid changes—exhibit every variety, from a mild

sporadic case to an epidemic of the most virulent type.

The following description applies to a moderately severe acute case. Irregular action of the bowels, griping pains, lassitude, and anorexia, generally precede the special symptoms for a few days. The actual attack is ushered in by shivering or rigors, with fever. Griping pains (*tormina*) are felt in the abdomen, with a strong desire to evacuate the bowels. The stools are at first fæcal and abundant; but they soon lose this character, becoming scanty, bloody, slimy from the presence of mucus, and emitting a most offensive odour. Still later, they become profuse, thin, watery, and dirty-looking, and perhaps contain fragments of sloughy mucous membrane, or dark coloured particles of blood. Profuse hæmorrhage sometimes occurs about this period. The desire to defæcate now becomes almost constant, and is attended with much tenesmus, irritation of the anus, and sometimes with very frequent micturition and dysuria. The tongue is furred, and complaint is made of thirst, loss of appetite, and occasionally of nausea and vomiting. The abdomen is tender, but not markedly tympanitic. There is always more or less febrile disturbance of a depressing type, which varies in amount and duration according to the extent and severity of the local mischief.

In favourable cases improvement begins about the end of the first week, but convalescence is usually protracted. When a fatal termination threatens, the symptoms assume an adynamic or typhoid character, with great prostration, a dry brown tongue, hiccough and vomiting, and more or less tympanitis, the patient finally becoming comatose. In the event of hæmorrhage, perforation, or the formation of abscesses in the liver, the symptoms preceding death will vary accordingly.

In other cases the disease may become *chronic* and

continue for an indefinite time. The action of the bowels is irregular; the stools are offensive, and exhibit from time to time a mucous, bloody, or serous appearance, according to circumstances. The general health suffers, as indicated by anæmia, loss of flesh, and sallow complexion.

DIAGNOSIS.—The symptoms and the characters of the stools as described may characterise any acute inflammation of the colon; and only a careful analysis of the circumstances under which it has arisen, coupled (when possible) with bacteriological investigation, will enable an opinion to be given as to its etiological nature and origin.

PROGNOSIS.—Sporadic dysentery is not nearly so fatal as the epidemic form. Much depends too on the type of the epidemic, the age and general condition of the patient, the presence or absence of complications, and the mode of treatment adopted. Very chronic cases are extremely troublesome.

TREATMENT.—In the first place it is necessary to remove the patient to cool, clean, and healthy quarters; and to insist upon rest in bed. In the early stages, much has been said in favour of magnesium sulphate in drachm doses, every one or two hours, until the motions have become fæculent; followed by a course of aromatic sulphuric acid and opium until the diarrhœa ceases. In tropical dysentery, ipecacuanha, in a dose of gr. 20 to 30, has still good reputation. It should be given on an empty stomach, and preceded by a subcutaneous injection of morphia. Absolute rest should be observed, and no fluid (beyond a little ice to relieve thirst) should be taken for some three hours after. It may be repeated at intervals of eight to ten hours if necessary. The diet should consist of rice-water, diluted gruel, weak broth, and the like; with white wine whey, according to the usual indications for alcohol. Milk is better avoided in the first instance. Hot fomentations should be applied to the abdomen. In milder cases, large

doses of bismuth sub-nitrate (gr. 20 to 30 every two hours), and minute doses of corrosive sublimate (gr. $\frac{1}{100}$ every hour) have been recommended.

Local treatment is of great value in chronic cases. The colon should be washed out as thoroughly as possible with enemata of hot water rendered antiseptic with borax, boric acid, or lysol 1 per cent., followed by an opiate—either in the form of a suppository, or a hypodermic injection of morphia. A short course of ipecacuanha (gr. iij.-v., every three hours) has been advised. Much benefit is often derived from tincture of perchloride of iron in full doses, coupled with gr. v. of Dover's powder night and morning. The regulation of the diet is of great importance, which should be light and nutritious. Manson says that a liberal diet of solid food often answers better than slops. Warmth is essential, and woollen clothing should be worn next the skin. Change of air, especially a sea voyage, is often of great advantage.

HYDROPHOBIA.

Hydrophobia is a specific disease, induced by direct inoculation of saliva from an animal afflicted with "rabies" or "madness," usually from the bite of a mad dog or cat.

The original wound generally heals readily, and the actual disease only develops after an interval of incubation, which is remarkable for its long and uncertain duration. The usual length of this period is from one to two months; but it has been known to vary from three or four weeks to as many months or even years. Neither sex nor age has any influence in its occurrence.

The disease begins with general malaise, depression of spirits, sleeplessness, and more or less restless anxiety. There may be uncomfortable sensations or actual pain at the seat of the primary wound, but it is a purely subjective

condition, and is probably induced by persistent attention to the part. The first really characteristic symptom is a sense of discomfort in the throat, with paroxysmal choking sensation on any attempt to swallow liquids, accompanied by a short sharp inspiratory gasp, said to resemble that caused by a sudden affusion of cold water. This respiratory spasm soon becomes more violent, till it involves all the extraordinary muscles of inspiration. The angles of the mouth are drawn outward, giving a curious sardonic expression. The spasm also becomes more readily excited, till at length it is induced by any sudden external impression, such as the mere sight of water, a draught of cold air, a loud noise, etc. The saliva cannot be swallowed, and the patient tosses about and spits wildly to get rid of it.

From this point the disease quickly develops. On the one hand the spasmodic tendency becomes more and more widely distributed, until general tetanoid convulsions are induced over and above the respiratory spasm, under the influence of stimuli such as those already mentioned. On the other hand, the general distress deepens, merging into delirium, or maniacal frenzy, with delusions and hallucinations. The face is flushed, there is moderate fever, little or no food can be taken, vomiting is not uncommon, and the patient quickly passes into a state of profound exhaustion. In some cases the symptoms exhibit a remarkable abatement towards the last, but the fact affords no justification of more hopeful prognosis.

The usual duration is from one to four days. Death is almost invariable, and generally occurs from asthenia. Sometimes, however, it occurs suddenly during a paroxysm—from asphyxia. With regard to *diagnosis* the one characteristic symptom is respiratory spasm, induced by attempts to swallow, and increasing in violence until it merges into convulsion.

The morbid anatomy is not characteristic. Gowers describes an accumulation of leucocytes around the vessels, with infiltration into the adjacent tissue; this change being either confined to, or more intense in, the medulla, contiguous to the lower part of the fourth ventricle.

TREATMENT.—As soon after the bite as possible a tight ligature should be applied above the wound, and the latter should then be well sucked or washed with warm soap and water, and afterwards thoroughly treated with some disinfectant fluid. Beyond temporary relief by the subcutaneous injection of morphia, or inhalation of chloroform, nothing can be done medicinally. The method of preventive inoculation devised by Pasteur is now generally accepted; and when adopted sufficiently early has been the means of saving many lives.

SAPRÆMIA—SEPTICÆMIA—PYÆMIA.

From a pathological standpoint, cases of blood-poisoning may be ranged in three groups: *sapræmia*, characterised by a simple intoxication of the system with the poisonous products of bacterial activity, causing disturbance which is directly proportionate to the amount of poison absorbed, and ceasing as soon as the source of supply fails; *septicæmia*, characterised by the presence, growth, and development of septic micro-organisms in the blood-stream; and *pyæmia*, characterised by multiple abscesses, resulting from septic emboli. The distinctions thus expressed have considerable practical value, though it is difficult or impossible always to maintain them at the bedside.

In an extended sense, blood-poisoning might be made to include all infective disease; but it is here restricted to that which arises in connection either (1) with the accidental introduction and absorption of the products of purely saprophytic and putrefactive organisms, in the form of

toxins or enzymes ; or (2) with the development within the body of facultative pathogenic organisms, such as staphylococci, streptococci, and *b. coli communis*, which are specially concerned with suppuration. The latter are often associated with other pathogenic organisms, to the manifestations of which they impart septicæmic characters.

The possible sources of blood-poisoning are various. It may proceed from dissection or post-mortem wounds ; from decomposition of blood-clot, damaged tissue, or the contents of the alimentary canal or bladder ; from the absorption of suppurative toxins, especially when associated with the decomposition of pus ; in connection with ulceration of any part of the body-surfaces—external or internal ; or from an accumulation of toxins produced *within* the body under physiological conditions, as in fatigue fever brought about by excessive muscular exertion.

Very little is known as to the exact conditions which determine the relative malignancy of septic organisms. They are probably partly external, and partly internal. No doubt there is an intimate relation to overcrowding, and to dirty and insanitary general surroundings. Owing to the better ventilation of hospitals, the much greater cubic space allotted to each patient, and the greater attention paid to cleanliness than formerly, the occurrence of the severer forms of blood-poisoning, such as pyæmia, has become rare ; and when they do occur are contracted under conditions in which these precautions are not or cannot be brought into operation. Again, the bodily nutrition and vitality of the patient count for much. Nervous depression, feeble health, alcoholic excess, inanition, and the like, lower the powers of resistance, and create a special vulnerability to septic invasion.

MORBID ANATOMY.—In *sapræmia* there are no obvious changes ; and in *septicæmia* there is nothing which is characteristic. Decomposition sets in earlier than usual ;

the blood is dark coloured, and coagulates imperfectly; and the internal organs are congested and softened. Abscesses are not uncommon. In *pyæmia* we may meet with phlebitis and secondary septic thrombosis in the neighbourhood of the primary infection, but the most characteristic feature consists of multiple abscesses. They are found most frequently in the lungs; but often also in the liver, spleen, kidneys, and other parts. They are undoubtedly of embolic origin; and the suppuration which follows immediately depends upon their infective nature. Suppuration may also take place in the subcutaneous tissue forming abscesses, and also in the joints, pleura, or pericardium.

In the blood there is usually a marked leucocytosis, chiefly affecting the polynuclear cells; while the red cells are more or less reduced, as the result of hæmolysis. Koch has called attention to the formation of little particles by the aggregation of septic organisms, entangled with blood-cells. The fact is interesting, as suggesting a possible source of emboli, other than detached thrombotic fragments.

SYMPTOMS.—There is sufficient general similarity between the symptoms of all forms of blood-poisoning to justify their being described together. All degrees of severity and duration may be met with. The onset in *sapræmia* is rapid as a rule, while in *septicæmia* and *pyæmia* it is usually more gradual. The onset is commonly attended by chilliness, or actual rigor, which may be repeated at irregular intervals. This is followed, in a typical case, by fever of a remittent or intermittent type; profuse sweating, alternating with a hot dry skin; a frequent feeble pulse; a parched tongue, with a tendency to sordes on the teeth and lips; loss of appetite, thirst, occasional nausea or vomiting, with irregular action of the bowels; marked prostration, with headache, restlessness, and more

or less wandering or actual delirium, which may merge into coma and collapse. The complexion is sallow, the cheeks are often flushed, and (in severe cases) there may be distinct jaundice. Dusky erythematous, petechial, or pustular eruptions may be observed. The spleen is frequently enlarged. More or less congestion of the lungs is commonly present, giving rise to cough, expectoration, and pains in the chest. Special symptoms may occur in connection with multiple abscesses, which vary according to their distribution. It should be noted that the temperature is often misleading, since it may be normal or sub-normal, even in serious cases.

The *prognosis* is extremely unfavourable, and death occurs either from asthenia or some secondary visceral inflammation.

Pyæmia is not always of this acute nature. Sometimes it runs a chronic course, the symptoms are less marked, and the fever is of an irregular type. This fact should especially be borne in mind, in connection with a pyæmic condition of great interest to the physician, arising from an infective form of endocarditis, of which something will be said in another place.

The indications for the *treatment* of these conditions are few and of a very general kind. When the lesion constituting the source of infection can be got at, practical measures should be adopted to counteract putrefaction, to provide an escape for pus which may have formed, and to promote a more healthy condition of the part. The patient must be well supported by nutritious food and stimulants; while the various excretions should be encouraged. Large doses of quinine and salicylic acid have been recommended, on account of their supposed antiseptic effects.

TETANUS.

Tetanus or lock-jaw is an acute specific disease, inoculable, and occasionally epidemic. It is due to absorption of a toxin, produced by a definite bacillus (discovered by Nicolaier in 1885, and found in earth or garden mould) which is limited to the seat of inoculation and is not found in the blood or tissues. Kitisato has succeeded in making pure cultures of this bacillus, subsequent inoculation of which produced tetanus. The disease usually occurs in connection with a traumatic injury, especially when accompanied with contusion. It is more common in hot than in temperate climates, and in coloured than in white races. Occasionally no obvious cause can be discovered (*idiopathic tetanus*). No characteristic lesions are found after death. Tetanus sometimes occurs in infants (*T. neonatorum*) as the result of infection of the umbilical cord.

SYMPTOMS.—A few days after the primary injury, the disease sets in with stiffness of the neck and jaws, which gradually develops into tonic spasm of these parts, causing *trismus*, or lock-jaw. The angles of the mouth are drawn out, giving a peculiar grinning expression (*risus sardonicus*). Occasionally the spasm is limited to these parts; but more often it extends to the muscles of the trunk, and (in a less degree) to the limbs. At this point, paroxysms of increased tonic spasm are observed, in the course of which the head is thrown back, the spine is arched backwards (*opisthotonos*), and the entire body and limbs become perfectly stiff. More rarely, the body is bent forwards (*emprosthotonos*), or to one side (*pleurosthotonos*). In very severe attacks the breathing is arrested by spasm of the respiratory muscles. The paroxysms usually only last for one or two seconds, and the patient then lapses into relative stiffness; but they recur on the slightest provocation, becoming more frequent as the

case proceeds. The attacks are acutely painful, and the patient often sweats profusely. The behaviour of the temperature varies. Sometimes it is normal or only a little raised ; but in other cases it rises to a high grade, especially on the approach of death. Until recently, the disease was generally fatal within seven days, from asphyxia or exhaustion ; but since anti-toxic treatment has been adopted, recovery has followed in a large proportion of cases.

DIAGNOSIS.—*Trismus* sometimes occurs from exposure to cold, or from reflex irritation (especially from the teeth), but it has no tendency to extend to other parts, and there are no paroxysms. In *strychnia poisoning*, the jaw is never involved early—if at all, and between the paroxysms the muscles are *relaxed*. *Trismus* and the absence of optic neuritis serve to distinguish tetanus from tonic convulsions of cephalic origin.

TREATMENT.—Absolute rest and quiet are necessary. Great attention should be paid to feeding ; and if food cannot be taken by the mouth, it must be given by enema or the nasal tube. Specific treatment by anti-toxic serum, derived from animals rendered insusceptible to the disease by graded inoculations, should always be adopted when available. The earlier the treatment is begun the greater hope of success. It should be injected subcutaneously once or twice a day, in doses of 10 c.c. Any wound should be cleansed antiseptically. Medicinal treatment is of subordinate importance. Morphia may be injected to relieve pain, as often as required ; and full doses of chloral have been recommended. Chloral hydrate in doses of a half-drachm or more, every four hours, has been strongly recommended, but its action requires to be carefully watched.

MALARIAL FEVERS.

Malarial, or marsh fever, is an acute specific disease, specially associated with low-lying, badly drained marshy districts. In its severer forms it is more common in the tropics, in parts of which it is often endemic. Malarial fever is now proved to depend primarily upon a protozoic amœboid micro-organism (*plasmodium malarie*) developing within the red blood-cells of the affected person. The disease is conveyed to man by a special variety of mosquito (*Anopheles*), which have themselves become infected by biting persons already afflicted. In the human body the parasite is first seen as a small nucleated rounded particle of protoplasm (*sporocyte*), occupying a red blood-cell, containing pigmentary granules of melanin derived from the hæmoglobin of the cell, and displaying amœboid movement. It afterwards undergoes segmentation into *spores*, which are subsequently discharged into the bloodstream (*sporulation*). Unless destroyed by treatment, the spores once more invade the red blood-cells, and the cycle is repeated. The duration of the cycle varies from about 36 to 72 hours, according to the species of parasite; and each act of sporulation gives rise to a paroxysm of fever. Such are the facts, in brief, of the asexual stage in the development of the parasite.

But besides the asexual sporocytes, we also meet with sexual forms (*gametocytes*), which circulate freely in the blood. In benign types of malaria they closely resemble mature sporocytes in appearance; but in malignant types they are *crescent-shaped*. Gametocytes undergo no change in human blood; but on being transferred to the body of the mosquito, the male gametocytes break up into flagellate bodies, which impregnate the females, and give rise to a series of changes which ultimately result in the formation of a number of filamentous spindle-shaped bodies, called

sporozoites. These enter the human body through the wound made by the bite of the mosquito, and set up infection.

Thus we see that the life-history of the malarial parasite includes two stages—an asexual stage which takes place in the human body, and a sexual stage in the bodies of mosquitoes. There is no doubt that the varieties of malarial fever correspond to different species of plasmodium, each species having a different rhythm of development.

Malaria is most prevalent in the spring and autumn months. No age or race is exempt; but negroes are less liable to infection than whites. All depressing influences, such as exposure to cold, want of food, alcoholism, and ill-health from any cause, predispose to its occurrence. There is great liability to recurrence, often after long intervals of freedom, and even after removal from an infected district.

MORBID ANATOMY.—The spleen is enlarged and dark-coloured. The enlargement at first subsides during the intervals, but later on it may become permanent from hyperplasia of the splenic substance. The liver also is enlarged and pigmented. The blood contains more or less black pigment, and the blood-corpuscles are diminished in number. The lymphatic glands and intestinal mucous membrane are congested and softened.

CLINICAL HISTORY.—Malarial fever presents many varieties, which may be conveniently arranged in two main groups: *benign* types and *malignant* types. It will be desirable first of all to describe a typical malarial paroxysm, or ague fit, such as occurs in ordinary benign cases. The attacks consist of febrile paroxysms, which recur periodically, and are separated by intervals of complete apyrexia. They either come on suddenly, or are preceded by a brief period of general malaise.

A paroxysm of ague comprises three distinct stages—a cold, a hot, and a sweating stage.

1. The *cold stage* begins with shivering, which soon passes into a severe rigor, accompanied with headache and pains in the back and limbs. The skin is dry and pale, the breathing is hurried, and the pulse is rapid, small, and irregular. The urine is pale and abundant. During the *whole* of this stage the temperature rises rapidly, reaching a high grade. The duration varies from about half an hour to one or two hours.

2. The *hot stage*, as a rule, sets in gradually, and the feeling of cold first gives place to one of pleasant warmth, but shortly afterwards the heat becomes intense. The skin is dry and pungently hot, the breathing is more rapid and deeper, while the pulse is full, strong, and frequent. The patient is excited and flushed, the thirst and headache are extreme, and sometimes there is more or less delirium. The urine becomes darker and of higher specific gravity, is intensely acid, and often contains a little albumen. The high temperature is maintained. The average duration is from one to four or five hours, but is often longer.

3. *Sweating stage*.—Profuse perspiration now sets in, and appears first on the face; a general sense of comfort supervenes, and the above symptoms subside. The quantity of urine diminishes, and it deposits urates on standing. The temperature rapidly falls, the patient drops into a refreshing sleep, and wakes relatively well. The duration is very variable.

The apyrexial period between one paroxysm and the next is called the *intermission* or *interval*, during which little or nothing is complained of.

1. **BENIGN MALARIA.**—The paroxysms are sharp and well defined. The attacks are often difficult to eradicate altogether; and though they may be temporarily arrested by treatment, they are prone to recur after a long time,

when the general health is depressed by any cause. Nevertheless, there is but little tendency to cachexia. It presents two varieties—*tertian* and *quartan*. In tertian fever, the attacks recur every 48 hours, and last about 11 hours. This is the commonest variety, and occurs in tropical and temperate climates alike. In quartan fever, the attacks recur every 72 hours, and last about 9 hours. It is relatively more common in temperate climates. In either variety there may be more than one generation of parasites. Thus in tertian fever there may be two generations sporulating on successive days, giving rise to an ague fit every day (*double tertian*). Similarly, in quartan fever—with two generations, there will be attacks on two successive days, followed by one day of freedom (*double quartan*); while with three generations, there will be an attack every day (*triple quartan*).

2. MALIGNANT OR ÆSTIVO-AUTUMNAL MALARIA.—This form is more common in tropical climates. The paroxysms, which may be quotidian, tertian, or irregular in rhythm, tend to merge into each other, and are separated only by *remissions* of temperature. Extreme anæmia develops, with a marked tendency to cachexia. Relapses are common. The onset is generally more gradual than in the benign form. The paroxysms are less defined, and last longer. Gastro-intestinal and nervous disturbances are more pronounced. Vomiting may be severe, and the vomit sometimes contains blood. The tongue is furred, and occasionally there is slight jaundice (bilious remittent). Headache is distressing, and sometimes ushers in low delirium. Cases vary widely in severity; some subside gradually at the end of eight to ten days, while others develop symptoms of a pernicious type and prove fatal.

Malignant malaria is liable to be associated with serious *pernicious symptoms*, owing to the tendency of the parasites to collect in various internal organs and structures. In

one form (*comatose*) we meet with severe nervous disturbance, which is sometimes preceded by acute violent delirium and culminates in coma. In another form (*algide*) there is severe gastro-intestinal disturbance, followed by collapse. In a third form (*hæmorrhagic*) there are hæmorrhages from various mucous surfaces; while, in all cases, malignant inflammatory complications are of common occurrence.

The term “malarial cachexia” is applied to a depraved condition of the general health, which persists after repeated attacks of malarial fever. It is characterised by a sallow complexion, languor, wasting, and various functional disorders, such as dyspepsia, neuralgia, or pains in the muscles and joints. Hæmorrhages are also sometimes met with. The spleen is usually enlarged, hard, and tender (*ague-cake*); and in some cases the liver is also increased in size.

DIAGNOSIS.—This is sometimes difficult, especially in irregular malignant types of the disease. Microscopic examination of the blood to detect the presence of the parasite is essential in such cases.

TREATMENT OF MALARIAL FEVERS.—*Preventive.* Those who live in malarial districts should avoid going out at night or in the early morning; should not sleep on the ground floor; should not drink the water of the district, unless previously boiled and filtered; and, finally, when attacked should remove (if possible) to some healthier locality without delay. It is well to take a daily dose of quinine (gr. ij.-v.).

During the paroxysm, the obvious indication in the cold stage is the application of warmth; while in the hot stage the body should be kept cool by sponging the skin with tepid water, free ventilation, and light clothing. Cooling drinks may also be given, and a little opium, if there be great restlessness.

During the intervals, quinine is our sheet-anchor, and

unquestionably exerts a powerful influence over the malarial poison. The drug must be given in large doses of 20 to 30 grains, either in one dose just before a paroxysm is expected, or in divided doses during the interval. If it cannot be retained, it may be given as an enema, or by *deep* hypodermic injection. It should be continued in smaller doses for at least a week or two after all the symptoms have disappeared. In æstivo-autumnal fever, the patient should remain in bed until all fever has ceased. Quinine should be given for a week in gr. v. doses (gradually reduced to gr. ij.) every four hours. The smaller dose should be continued in all cases, three times a day for at least three weeks after the pyrexia has ceased. It is worthy of notice that great importance has been attached to keeping the bowels properly open.

SYPHILIS.

Syphilis is a specific disease, which may be either acquired or inherited. In the former case it can only be communicated by direct contact with an infected person, which occurs for the most part during sexual intercourse. In the latter case it is transmitted from the parent to the offspring *in utero*. One attack of the *primary* disease would appear to confer protection against its repetition. Recent investigations undertaken by Schaudin and Hoffmann appear to have definitely established the association of syphilis with a specific protozoic organism, known as *spirochæta pallida*.

PRIMARY SYPHILIS.—After a period of incubation of about a month, a “primary sore” appears on the site of inoculation, which is termed a chancre. It begins as a hard flattened papula, which afterwards enlarges and breaks down, forming a superficial ulcer with an elevated margin and an indurated base. After about two months

it gradually heals and becomes absorbed. The nearest lymphatic glands are always somewhat enlarged and tender, but are freely movable, and have no tendency to suppurate. After the disappearance of the sore they usually soon regain their normal size, or nearly so ; but sometimes they remain more or less enlarged for months or even years.

SECONDARY SYPHILIS.—From six weeks to three months after the first appearance of the primary sore, some slight febrile disturbance, debility, and anæmia usher in a group of so-called *secondary symptoms*, which present considerable variety. The most important are as follows :—

1. *Cutaneous eruptions*, which may be macular, papular, vesicular, scaly, or pustular. They are mainly characterised by (a) a coppery colour ; (b) a brownish stain left for a considerable time after their disappearance ; (c) the occurrence of various types of eruption at the same time ; (d) a tendency to circular form and grouping ; (e) an absence of itching ; and (f) by an aptitude to affect parts such as the forehead, soles of the feet, and palms of the hands, which are rarely affected by non-specific eruptions. The hair is also liable to become dry and fall off.
2. *Lesions of mucous membranes*, including (a) superficial ulcers of the tonsils, soft palate, etc. ; and (b) mucous tubercles at the orifices of the mouth, nose, rectum, or elsewhere.
3. *Lesions of the eye*, including (a) iritis ; and (b) choroido-retinitis.
4. *Pains in the joints and bones*, due to slight periostitis.

TERTIARY SYPHILIS.—Unless the further progress of the disease be checked by appropriate treatment, a fresh series of lesions may occur (*tertiary syphilis*), after an interval which varies from one to several years. There is no sharp distinction between secondary symptoms on the one hand, and tertiary symptoms on the other ; but the latter are mainly characterised by their late appearance, irregular distribution, the tendency to involve a considerable thick-

ness of tissue with liability to deep ulceration, and frequent implication of viscera and other parts by the formation of gummata. The most important are as follows :—

The most characteristic feature of tertiary lesions is the formation of local tumours, called *gummata*, which at first consist of soft gelatinous granulation-like tissue, but afterwards becomes hard, with a tendency to cheesy necrosis at the centre, while the periphery undergoes fibroid change. Gummata occur most often in the periosteum, liver, testes, and brain. Closely analogous infiltration is met with in the skin and mucous membranes (soft palate, pharynx, or rectum), in which situations it readily breaks down, and leads to destructive or serpiginous ulceration. In other cases the walls of the arterioles are attacked, giving rise to an obliterative arteritis, which is a fertile cause of thrombosis. Another remote and probably indirect effect of syphilis is manifested by localised degeneration of nerve-tissue, associated with fibroid change (*sclerosis*).

Sooner or later, if the progress of the disease be still unchecked, the so-called syphilitic cachexia sets in, and the patient may finally die, either from exhaustion or from some intercurrent affection.

CONGENITAL SYPHILIS.—Symptoms usually first appear at the end of the third to the sixth week after birth in the form of cutaneous eruptions resembling those of the secondary period of acquired syphilis, and of most frequent occurrence on the nates, abdomen, palms, and soles. Congestion and swelling of the nasal mucous membrane, with increased secretion, also constitute common initial symptoms (*snuffles*). Mucous tubercles about the mouth or anus, and interstitial inflammation of the cornea, are frequently met with. The temporary incisor teeth are cut early and as a rule with little difficulty; but they soon decay, and the permanent teeth which replace them are ill-formed, short, peg-shaped, and present a central notch.

If the disease proceeds unchecked, lesions of the tertiary type may occur. The child's appearance is cachectic, the features pinched, the skin dry and shrivelled, and it gradually sinks. On the other hand, appropriate treatment by mercurials will speedily restore the child, in most cases, to a condition of comparative health, if begun sufficiently early.

TREATMENT.—The only effectual remedy for syphilis is mercury. It may be given either (a) by *inunction* with mercurial ointment; (b) by *fumigation* with the vapour of calomel; or (c) by *internal administration* of some mercurial preparation. For infants the method of inunction is the most convenient. For adults, in all ordinary cases, nothing answers better than gr. j. doses of mercury or chalk, three or four times a day, in the form of a pill made up with a little ext. gentian. The treatment should never be pushed to the extent of salivation, and should be continued for at least six months after the disappearance of symptoms, in smaller doses given less frequently. When more rapid effects are desired, the employment of fumigation, or of blue pill (gr. ij. to iij. *ter die*) with a little opium is preferable. Iodide of potassium is of special service in tertiary syphilitic affections of the internal organs, particularly of the nervous system. In such cases the dose should be free, and be rapidly increased. Above all it should be remembered that syphilis is a debilitating disease, and the importance of all treatment which tends to the improvement of the general health cannot be over-rated. As to the propriety of marriage on the part of those who have contracted syphilis, it is laid down by the best authorities that, after two years' complete freedom from all evidence of syphilis, marriage is practically safe.

Further details must be sought in surgical textbooks.

TUBERCULOSIS.

Tuberculosis is a specific infective disease which is due to the development within the body of a specific bacterial organism (*B. tuberculosis*). The organism in question consists of slender non-motile rods, about one-third of the diameter of a red blood-cell in length, and variously grouped.¹ They occur in all tuberculous lesions, and are cast off in vast numbers in the sputa of those who suffer from tuberculosis of the lung.

The bacilli enter the body in different ways—most frequently either by inhalation in the act of breathing, giving rise to tubercle of the air-passages and lungs; or by ingestion of tuberculised food, or by swallowing tubercular sputa, giving rise to tubercle of the bowel or mesenteric glands. Much more rarely, tuberculosis may be contracted by accidental inoculation.

In many cases, a local tuberculous lesion occurs primarily at the point at which the virus enters the system; but this is by no means necessary. For instance, in children infected with tubercle through their food, there is little doubt that the bacilli often pass through the intestinal wall and affect the mesenteric glands without setting up any intestinal lesion. Disease may subsequently spread to other parts by the lymphatics, by the blood-vessels, or by direct continuity of tissue. Or again, tubercle-bacilli may be carried from one part of the body to another through the natural passages—from the lungs, for instance, to the intestine, by swallowing infected sputum; or from the kidneys to the bladder, by the urine.

The two chief sources of infection are the sputa of persons suffering from pulmonary tuberculosis, and milk from

¹ The method of detecting tubercle bacilli in the sputa will be described in the Appendix.

tuberculous cows. Considering the frequency of the former disease, and the reckless way in which sputa are distributed both in and out-of-doors, together with the prevalence of bovine tuberculosis, there must be few who escape the introduction of tubercle-bacilli into their bodies at some time of life. Yet only a minority contract the disease, the majority enjoying immunity. It comes to this, that only those are attacked for the most part who are predisposed. Anything which reduces vitality predisposes to tuberculosis. Predisposition may be due to general debility from any cause, vicious habits such as alcoholism, inanition, or exhausting chronic disease such as diabetes. But predisposition may be inherited. In this connection, there are two inheritable types of constitution which are of importance. The one type (scrofulous or strumous) is characterised by slow nutrition, and general feeble powers of resistance on the part of the tissues, so that inflammatory damage is readily induced, and when started is apt to be unusually chronic and sluggish. Children of this type have usually a short stumpy figure, thick opaque skin, a round face, ugly features, coarse hair, a thick upper lip, a broad nose, dull temperament, and backward intellect. The bones, moreover, are thick, the teeth decay early, and the belly is tumid. The second type (sometimes called "tuberculous") is characterised by premature rapid growth out of proportion to nutrition, associated with a special instability of the body tissues, and a proneness to acute rapid inflammation from relatively trifling damage. Children of this type are often recognisable by a special build and appearance of the body, characterised by a tall, thin figure, transparent soft delicate skin, oval face, regular features, silky hair, large bright eyes with long eyelashes, excitable temperament, and general precocious development.

The characteristic anatomical features of tuberculosis

consist in the formation of so-called "tubercles" in the affected part. An elementary tubercle (or *grey granulation*, as it used to be called) is a minute, pearly-looking, greyish, non-vascular nodule when isolated, but larger nodules may result from fusion of adjacent tubercles. Tubercles may be found in the lungs, intestines, lymphatic glands, serous membranes, spleen, and other viscera. Microscopically a typical tubercle consists of a centrally situated many-nucleated giant cell, surrounded by a zone of largish cells of an epithelioid type, with oval nuclei, outside which is another zone of small lymphoid cells; the whole being embedded in a reticulum of doubtful nature. While this is the usual structure of a fully formed tubercle in its early stage, before it has begun to caseate, it cannot be regarded as absolutely characteristic. It is in the circumstances of their origin and evolution, rather than in their structure, that we should look for the specific characters of tubercles. Before long, caseation begins at the centre of each tubercle, gradually involving the whole of it, often converting a group of adjacent tubercles into a caseous mass of considerable size. Each tubercular focus gradually becomes enclosed by a zone of fibroid tissue, reactive in its origin.

The further progress varies. 1. The nodule may soften, and become converted into a puriform liquid, forming an ulcer when superficial, or pseudo-abscess when situated more deeply. 2. It may calcify, becoming quiescent and inert. 3. It may undergo fibroid organisation, and shrink up.

A tubercle must be considered as a local new formation, occurring as a direct result of irritation set up by tubercle-bacilli which have found their way to the part. Whether its cellular elements are derived from fixed tissue cells, or from leucocytes attracted to the focus of irritation, is not as yet definitely settled. The surrounding tissues also become involved in a diffuse secondary inflammation,

which varies widely in character and extent according to circumstances.

MILIARY TUBERCULOSIS.

The term *miliary tuberculosis* is applied to acute cases in which a more or less disseminated eruption of grey tubercles takes place in various organs and structures of the body, attended with well-marked generalised infection. It always implies an invasion of the blood-stream by tubercle-bacilli. This may occur either by direct implication of the blood-vessels; or indirectly, as by tuberculous disease of the thoracic duct. The parts most commonly affected are the lungs, liver, spleen, kidneys, pleura, peritoneum, and cerebral membranes, but the distribution is unequal and varies in different cases, the lungs being most often attacked. The source of infection is almost always to be found in some pre-existing focus of chronic tubercular mischief, especially in the lungs and lymphatic glands; but occasionally the disease appears to be primary. It is most common in early life.

SYMPTOMS.—It should be understood that the symptoms vary considerably in different cases, according to the distribution of the lesions, and virulence of the infection, though a broad similarity may be recognised. The invasion is generally gradual, and marked by progressive weakness, headache, anorexia, and fever. Sometimes, however, it is sudden, and attended with rigors, nausea, and vomiting. There is usually some bronchitis; the pulse is frequent and feeble, but seldom dicrotic; and the general condition closely resembles typhoid fever, especially if the bowels happen to be somewhat relaxed, and distinction is often a matter of extreme difficulty. The chief points in favour of tuberculosis are marked irregularity of the temperature, which is of a remittent or intermittent type; a certain duskiness of complexion; greater frequency of

the breathing; more frequent constipation; and the absence of rash. As the disease progresses, the emaciation and prostration increase, the patient finally becomes comatose, and dies after an illness of four to ten weeks.

Sometimes the lung symptoms are specially pronounced, and we meet with marked cyanosis, cough, extreme dyspnoea, and expectoration, with abundant moist sounds over the whole of the chest. The sputa are not characteristic, and more often do not reveal tubercle bacilli on examination. In other cases again prominent nervous symptoms are observed. The spleen is usually somewhat enlarged. Tubercles may sometimes be found in the choroid, by means of the ophthalmoscope.

The chief diseases with which miliary tuberculosis may be confounded are typhoid fever, acute capillary bronchitis, and infective endocarditis.

The *treatment* is unfortunately purely palliative, and a fatal issue is probably invariable.

The local forms of tuberculosis will be described in later chapters.

ANTHRAX.

(SPLENIC FEVER. WOOLSORTER'S DISEASE.)

THIS is a specific disease due to a special bacillus which affects various animals and may be communicated from them to human beings. It is most common among tanners, wool and hide sorters, and furriers, who are liable to handle the skins of affected animals.

As an *external* disease (*malignant pustule*) inoculation occurs through some scratch or abrasion of the skin. In a short time a pimple appears which quickly becomes vesicular, bursts, and finally forms an eschar resting on a hard red base. The tissue around is cedematous, and the adjacent glands are enlarged. Sometimes a malignant cedema occurs without a sore. The amount of constitutional disturbance varies, but if untreated it tends to a fatal issue in the course of a week or earlier.

As an *internal* disease a gastro-intestinal and a pulmonary variety are met with, according as the contagium gains access to the body by the alimentary or respiratory tract. In the former, the symptoms set in with abdominal pain, vomiting, and diarrhœa; while in the latter they are characterised by thoracic oppression, dyspnœa, moderate cough, and sometimes bloody expectoration. In both, symptoms of acute blood-poisoning occur, with fever and intense prostration, frequently ending in fatal collapse.

Malignant pustule should be treated by excision, and subsequent application of antiseptics. Considerable progress has of late been made in the treatment of anthrax by the injection of serum of immunised animals, but it is not yet perfected.

ACTINOMYCOSIS.

THIS also is a specific disease common amongst cattle, and communicable from them to man, probably by means of food as a rule. It depends upon a fungus (*actinomyces*), occurring in the form of granules, made up of radiated threads with bulbous terminations. On its entry into the body, it adheres to some part of the respiratory or alimentary mucous membrane, there setting up little inflammatory tumours, which suppurate. Larger tumours may be formed by an aggregation of small nodules. In some cases it affects the mouth or tongue, and others have been recorded in which the intestines were primarily implicated. It is also met with in the lungs, setting up bronchitis and broncho-pneumonia with putrid expectoration. Secondary deposits in other parts are not uncommon. The symptoms are those of chronic pyæmia, and the termination is usually fatal; but when the growth is accessible to surgical treatment, recovery has followed on complete removal of the growth or on thoroughly scraping out the resulting abscess-cavities.

PLAGUE.

PLAGUE is an acute contagious disease, characterised by bubonic enlargement of lymphatic glands, and sometimes by carbuncles, dependent upon a specific bacillus discovered by Kitasato. Infection occurs chiefly by contact with the diseased person or with contaminated clothing, etc. Lower animals, especially

rats and mice, are largely in some way responsible for the spread of the disease. Overcrowding, neglect of the person, and insanitation are potent predisposing conditions. It is one of the most fatal of human diseases.

Although met with in varying degrees of severity, the symptoms of the ordinary form are as follow :—After an incubation of 3 to 6 days, it begins with restlessness, headache, pains in the back and limbs, and great depression. The temperature soon afterwards rises rapidly, with a quick pulse, and hurried breathing, and in severe cases the patient becomes collapsed, and may die forthwith. If he survives, the lymphatic glands (generally those of the groin) become enlarged and tender; the other glands quickly follow suit, and often suppurate. Carbuncles also appear in various parts. About this time petechiæ are apt to occur, together with hæmorrhages from the mucous membranes, under which the patient more often sinks.

As a preventive measure inoculation, with increasing doses of a sterilised bouillon culture of the plague-organism has met with encouraging results. As to treatment of the developed disease, little can be done beyond alcoholic stimulation, good nourishment, and hygienic precautions.

LEPROSY.

LEPROSY is a chronic contagious disease due to a specific bacillus, characterised sometimes by nodular infiltrations of the skin (*tubercular leprosy*), and sometimes by analogous affection of the nerves (*smooth or anæsthetic leprosy*). In their later developments, both forms are commonly combined. It is endemic in Norway, East and West Indies, and various parts of Asia, Africa, and America. The conditions of its development and communication are very imperfectly understood. Hereditary transmission has not been proved.

The tubercular form usually begins with the appearance of reddish sensitive patches on some part of the face, extending later to the trunk and limbs. These may disappear after a time. Later on, either on the site of such patches, or elsewhere, but chiefly on the face, causing great deformity of expression, hard nodular infiltrations appear. The mucous membranes of the mouth, nose, or larynx, may be also affected at a still later stage. These nodules may persist for an indefinite time, or

wither away, or ulcerate, discharging a scanty thin pus. The eyelashes, eyebrows, and hairs on the face fall out.

The smooth or anæsthetic form begins with pains in the limbs, and areas of numbness or actual anæsthesia, sometimes (but not necessarily) associated with maculæ, as above described. The superficial nerve-trunks may be felt to be irregularly thickened. Trophic troubles, usually in the form of unhealthy destructive ulcerations, are of common occurrence, especially on the hands and feet, and sometimes leading to actual loss of fingers and toes.

With a variable course, sufferers from leprosy drag out their miserable lives for 5 to 15 years, until finally released by tuberculosis, blood-poisoning, or some other intercurrent disease.

Leprous infiltrations, whether of skin or nerves, consist of nucleated cells, embedded in a matrix of fine connective-tissue, and enclosing myriads of leprosy bacilli, closely resembling those of tuberculosis.

The disease is incurable.

BLACKWATER OR HÆMOGLOBINURIC FEVER.

THIS is a specific disease, which only occurs in those who have previously suffered from malaria; but its precise nature is uncertain. It is characterised by marked hæmolysis, with liberation of hæmoglobin in the plasma of the blood, and subsequent elimination in the urine, causing hæmoglobinuria. Its onset is abrupt, attended with rigors, and followed by high fever. The chief symptoms are pains in the back, limbs, and abdomen; much prostration and frequent vomiting; marked jaundice and frequent micturition, with the passage of dark-coloured urine. In about 4 to 6 hours, the temperature falls, the patient breaks out in a profuse sweat, and the paroxysm is over. The attacks are very apt to recur; and, in severe cases, may be repeated for several days almost without intermission.

The urine is dark or porter-coloured, loaded with albumen, depositing a dark red sediment, with many casts, but few blood-cells. This condition may be intermittent, coinciding with the paroxysms; but in severe cases it is practically continuous.

Some cases are mild and recover quickly; others are severe, and recover very slowly; while a varying proportion end fatally with suppression of urine, and symptoms of an adynamic type. Little or nothing can be done for it beyond the relief of symptoms as they arise. General experience is against giving quinine.

MALTA FEVER.

MALTA FEVER is a specific disease, characterised by recurrent attacks of pyrexia, variable in type and duration, often extending over long periods. It is caused by a micrococcus, known as *M. melitensis*. It was formerly specially prevalent in Malta (hence its name), but since the organism has been traced to the milk supply from native goats, and steps have been taken to correct this, there is every probability that the disease will be stamped out in the island.

Its onset is slow and insidious, and is chiefly marked by fever, general malaise, great weakness, headache, restlessness, and muscular pains, with some cough and expectoration. The spleen is enlarged. The joints are painful, and often swollen from effusion within them. Orchitis is not uncommon, and anæmia is a marked feature. In the course of a week or two the temperature gradually falls, and the symptoms subside; but a few days later (or it may be longer) a relapse takes place, which may be repeated again and again over a long period. Notwithstanding its long duration, the mortality is singularly low. The chief disease from which it has to be distinguished is typhoid fever. No specific treatment is available.

DENGUE, OR DANDY FEVER.

DENGUE is a specific fever peculiar to the tropics, which occurs in the form of quickly spreading epidemics. Its cause is unknown. It begins suddenly with chilliness or rigor, and a rapid rise of temperature. The chief symptoms are severe pains all over the body, spreading to the joints, which become swollen; marked prostration; aching of the eyes, with suffusion of the conjunctivæ; a flushed and puffy face; often some sore throat; with the usual results of febrile disease. About the third day all these symptoms quickly subside, but leave the patient very weak and sore. Then, usually after an interval of two or three days, the fever *relapses*, with a return of the pains, in association with a rash (*terminal eruption*). The nature of the rash varies; but more often it is red and patchy, something like measles, usually appearing first in the palms of the hands. The secondary fever does not last long—about two or three days, as a rule—when recovery takes place. The disease, however, varies widely in its severity and in its details. It is very rarely fatal, but con-

valescence is often very tedious, with troublesome recurring rheumatic pains in the joints. The treatment is purely symptomatic. The salicylates, phenacetin, or morphia may be given to relieve the pains.

BERI-BERI.

BERI-BERI is a specific form of multiple neuritis, chiefly occurring in warm moist climates. It is endemic in many parts of the East. Although believed to be microbic, no special organism has yet been identified with it. Some believe that it is due to eating diseased rice. The disease begins gradually with progressive weakness of the limbs, especially the legs, followed by wasting, marked tenderness of the affected muscles, and more or less cedema. A somewhat characteristic feature is irregularity of the heart, with a liability to sudden attacks of cardiac dilatation. The course is slow, and the mortality varies widely. In temperate climates the prognosis is good under favourable conditions; but it is often fatal when the conditions are less favourable from implication of the cardiac nerves, syncope, or some intercurrent complication.

The main indications for *treatment* are: Removal (when possible) from endemic areas, absolute rest in bed, nutritious food, avoidance of overcrowding, and good hygiene. Strychnia and arsenic internally, together with massage and faradism, are beneficial for the paralysis.

SECTION III.

GENERAL DISEASES.

RHEUMATISM.

GENERAL CONSIDERATIONS.—There are few morbid conditions the nosological relations of which are in a more confused state than those which are called *rheumatic*. The term "*rheumatism*" is vaguely applied in common speech to a group of painful inflammatory affections of the joints, or the structures in connection with them—muscles, tendons, ligaments, and so forth, occurring as a manifestation of some general disorder or disease. From this general conception of rheumatism, gout has long been separated, by reason of its special association with deposits of bi-urate of soda. Also by clinical observation alone we have been enabled to differentiate, with more or less confidence, certain acute types of arthritic disease, such as rheumatic fever; acute arthritis occurring in connection with specific fevers, septicæmia, and gonorrhœa; and perhaps one might add the acuter forms of rheumatoid arthritis. It is, however, when we come to the sub-acute and chronic forms of arthritic disease that the difficulties of differentiation and classification are so great; and it is not likely that much progress will be made in this direction, until we know more of the exact pathological conditions under which this class of disease (as a whole) arises.

It is highly probable that in every type of general arthritic disease we have to deal with the circulation in the blood and lymph of some material of an irritating nature, which is liable to set up inflammatory changes in various tissues and structures of the body, especially the joints, muscles, and fibrous tissues. Two alternative modes of origin suggest themselves—namely, metabolic and microbic. The irritative material, that is to say, may result from some disturbance of metabolism, or be the product of microbic activity of some kind. Infective arthritis is undoubtedly microbic in origin, and recent research seems to point to an analogous cause of rheumatic fever. On the other hand true gout is almost certainly a metabolic disorder; and, if so, the fact at least bears witness to the power of disorder of this kind to set up joint inflammation, though in this case it is indirectly brought about by uratic deposits in the joint. Whether rheumatoid arthritis and other sub-acute and chronic joint affections are due to microbes or not is at present unknown. In any case too much diagnostic importance should not be attached to variations in joint-features. Such differences mainly depend upon differences in the intensity and duration of the inflammation. There is no joint-change, which may not be present, under certain conditions, in almost every type of general arthritic disease—especially in its chronic forms.

It must be understood therefore that the classification adopted in the sections which follow makes no claim to pathological accuracy, which in the present state of knowledge is impossible; but it fairly corresponds with what clinical observation and practical convenience indicate.

ACUTE RHEUMATISM.

The essential nature of acute rheumatism, or rheumatic

fever, is still doubtful ; but there is a growing disposition to attribute it to a specific micro-organism ; and Poynton and Paine have isolated a diplococcus which they regard as the true cause of the disease. There is some evidence of hereditary predisposition, and there is little doubt that rheumatism is favoured by exposure to cold and damp, by over-exertion, and by conditions attended with fatigue or depressed general health. The disease is most common in young adults, especially between fifteen and thirty years of age.

MORBID ANATOMY.—The changes as observed in the joints are characterised by an acute superficial inflammation of the lining synovial membrane, which becomes congested, and loses its natural polish. An effusion into the joint-cavity usually follows, which consists of clear or slightly turbid serosity, with flakes of lymph in suspension. More often, these changes rapidly subside, and the joint recovers its normal state. After repeated or very prolonged attacks, however, persistent thickening of the joint-structures is apt to take place. Similar changes may affect the sheaths of tendons, and the synovial bursæ. There may be also a little œdema of the tissues around the joint. Small painless subcutaneous fibroid nodules are sometimes met with, especially in children, in the neighbourhood of the joints, and over bony prominences elsewhere. With the exception of these nodules there is nothing characteristic about rheumatic inflammations.

SYMPTOMS.—The actual attack is ordinarily characterised by inflammation of the joints, fever, profuse sweating, and anæmia, and may begin abruptly, or be preceded for a few days by wandering pains in the joints and muscles with more or less malaise, or by an attack of tonsilitis. The joints usually affected are of middle size—viz. the knees, ankles, elbows, and wrists. They are extremely painful (the pain being intensified by the least movement), tender,

swollen, and hot. The swelling is partly due to fluid effusion within the joint, and partly to œdema of the tissues around it. The skin over the inflamed joints may be slightly reddened. The number of joints attacked at one time varies greatly; and there is a tendency for the trouble to migrate suddenly from one joint to another, which is characteristic. Similar changes may be observed in connection with the sheaths of certain tendons, or synovial bursæ.

The amount of *fever* varies much, but it rarely exceeds 104° in uncomplicated cases. It is characterised by considerable irregularities; and its progress and duration also vary considerably according to the behaviour of the joint-inflammation, the occurrence of complications, and the treatment adopted. *Profuse perspiration* with a sour smell, of very acid reaction, and often accompanied with sweat rashes (sudamina and miliaria), is almost invariable. Notable *anæmia* is another common feature, the intensity of which is proportionate to the severity and duration of the disease. It depends upon rapid destruction of red blood-cells, accompanied with a well-marked leucocytosis. The *urine* is scanty, high-coloured, very acid, and deposits urates in abundance. The bowels are usually constipated. Delirium is rarely met with, except in connection with some inflammatory complication such as pericarditis, with previous alcoholism, or as a premonitory symptom of hyperpyrexia.

Next to arthritis, cardiac inflammation is by far the most frequent and most important manifestation of acute rheumatism, giving rise (in the order of their relative frequency) to endocarditis, myocarditis, and pericarditis. Among the less common rheumatic inflammations we meet with pneumonia, pleurisy, and meningitis. Rheumatic subjects are also prone to tonsillitis, chorea, and erythematous eruptions. Hyperpyrexia is occasionally observed, but the

conditions on which it depends are by no means clear. It may be preceded by delirium and drying up of the skin ; and if not relieved by prompt treatment, leads on to a fatal coma. It is a noteworthy fact that, in young children, articular inflammation is much less common than in adults ; while, on the other hand, cardiac and serous inflammations are much more frequent. Rheumatic nodules also seldom occur except in children.

The duration of rheumatic fever varies greatly—partly according to the intensity of the process, and partly according to the nature of associated inflammations. A simple uncomplicated case of the articular type, if left to itself, may last for several weeks ; but with prompt and appropriate treatment the pains and fever usually subside in a few days. If, however, the treatment be suspended prematurely, relapse is almost certain to occur ; and sometimes convalescence is delayed for a long time, by the persistence of inflammatory trouble in one or more joints. Uncomplicated articular rheumatism is seldom or never directly fatal ; but the liability to recurrence, and the fact that each attack is attended with the risk of serious complications, invest it with great importance.

The *diagnosis* from septic arthritis and gout will be considered with the description of those diseases.

TREATMENT.—Under all circumstances rest in bed should be insisted upon, and the joints should be wrapped in cotton wool. The diet should be liberal, though plain and light ; alcoholic stimulants are rarely required.

Many medicinal remedies have been from time to time recommended, but treatment by the salicylates of ammonia, soda, or potash, is generally recognised as the best method, though it is not truly specific. The salicyl compounds appear to produce their effects, not upon the rheumatic process itself, but upon some of its immediate products, by which the latter are in some way deprived of their power

to set up inflammatory damage. On such damage, when it has once taken place, the salicylates have no influence. The salicylate should be given in doses of gr. x. to xx. (often advantageously combined with sodium bicarbonate) every two or three hours until the pains subside and the temperature has returned to the normal level; but afterwards at longer intervals. It is readily taken, and unpleasant symptoms rarely occur. Occasionally, however, there may be some headache, buzzing in the ears, deafness, and slight mental confusion, but these symptoms quickly subside on suspending the use of the drug. The remedy should be continued for at least ten days or a fortnight after the cessation of acute symptoms, or relapse of trouble will take place. The secretions must be attended to; and great comfort is sometimes derived from sponging the body with tepid water, of course avoiding all unnecessary exposure.

The local application of poppy fomentations, or light poultices, sometimes gives relief to the pain; and in specially severe cases the hypodermic use of morphia may be justified. The cautious application of blisters to the neighbourhood of a *persistently* painful joint may be of service. Hyperpyrexia calls for prompt antipyretic treatment, by repeated cold wet-packs; or better still, when possible, by immersion in a tepid bath, gradually cooled down to about 75°. The patient should be removed from the bath as soon as the temperature in the mouth or rectum falls to 101°, and be returned to bed between blankets. The bath may be repeated as occasion requires.

Care should be taken during convalescence to avoid exposure to cold or damp. Quinine in combination with arsenic is a useful tonic at this stage.

CHRONIC RHEUMATISM.

Chronic rheumatism is sometimes a sequel of the acute

form, but more often it is chronic from the outset, especially in those who are engaged in laborious work, with exposure to cold, damp, and privation. Yet again, it may take the form of recurrent sub-acute attacks, affecting different joints from time to time. It is most frequent after forty years of age, but earlier ages are not exempt. There may be no apparent structural changes in the joints, but more often the fibrous structures around the joints are more or less thickened. Synovial effusion is exceptional, and the bones and cartilages are rarely affected. The muscles inserted around the joint are prone to atrophy, partly from disuse, and partly from associated neuritis.

The chief *symptoms* are pain, tenderness, and stiffness in one or more joints; worse in cold and damp weather, and generally worse at night. It is also worse as a rule after rest, and better after moderate use of the joint. The movements of the joint are painful and restricted, but true ankylosis does not occur. There may be creaking on passive movement, but no grating. The general health is usually impaired, and anæmia is common, together with a liability to neuralgia and muscular rheumatism.

TREATMENT.—The salicylates, in combination with alkaline bicarbonates and iodides, are sometimes useful. Arsenic and strychnia are valuable where there is marked debility. The diet should be nutritious and readily digestible. As a rule more benefit is derived from judicious local treatment by friction and manipulation of the joints, with anodyne or stimulating liniments; by radiant heat; or by a course of baths at Harrogate, Bath, or Buxton. Warm clothing and protection from cold and damp are essential.

MUSCULAR RHEUMATISM—MYALGIA.

Under this term are included certain painful affections of the muscles, and the fibrous structures in connection with them. Nothing is definitely known as to the precise

nature of this affection. It occurs as the result of exposure to cold draughts, damp, and muscular strain of various kinds. It appears to be more common in subjects of a rheumatic or gouty habit, and is not infrequently associated with other forms of rheumatism. After repeated attacks, small indurated patches may be sometimes detected in the affected muscles, which are quite distinct from the subcutaneous nodules of acute rheumatism, the peri-articular nodosities of rheumatoid arthritis, and the bursal enlargements of chronic gout, and are suggestive of an interstitial myositis. But, as Osler points out, it is by no means proved that the seat of trouble is essentially muscular, and many regard the affection as neuralgic.

Special terms are applied to its varieties. Thus, it is called *pleurodynia*, when the intercostal and thoracic muscles are affected; *lumbago*, when the muscles of the loin are affected; *stiff neck*, when the sternomastoid and other muscles of the neck are involved. The last should be distinguished from spasmodic torticollis, which is due to other causes. The local symptoms are pain—greatly increased by movement, stiffness, and tenderness. In some cases there is a tendency to spasm of the affected muscles. No constitutional symptoms are present, as a rule, but there may be mild fever, and slight gastro-intestinal disturbance. The duration is very variable, and recurrence is common.

TREATMENT.—A hot air or vapour bath, followed by a brisk purge, is useful at the outset; and among internal remedies, alkalies, chloride of ammonium, alkaline salicylates, and iodide of potassium, are the best. Locally, pain may be relieved by hot fomentations or poultices, gentle friction with anodyne liniments, and firm strapping (in pleurodynia). Warm clothing is also of great importance.

INFECTIVE ARTHRITIS.

Under this term are included those cases of arthritis which occur in association with infective diseases, such as pyæmia, gonorrhœa, scarlet fever, pneumonia, and typhoid. There is little reason to doubt that it is due to the presence of microbic products within the joint-cavities—not necessarily derived from the specific organism of the primary disease, but possibly from some mixed or secondary infection. Special differences in the distribution and character of the joint affection are observed, according to the particular infection with which it is associated; but there are general features common to the group. Thus, the joint trouble for the most part occurs in the later stages or as a sequel of the disease which gives rise to it. At first there may be pain in several joints; but it soon settles down, as a rule, to one or two joints, in which the trouble persists for a long time. Considerable inflammatory damage occurs, with a tendency to fibrous ankylosis; and suppuration sometimes takes place, especially in pyæmic and scarlatinal varieties.

Gonorrhœal rheumatism is the best known and most important member of the group. It begins usually about three weeks after the onset of the infection. It may assume various forms. (1) Simple arthralgia, without structural change. (2) A poly-articular inflammation, which either passes slowly off, without permanent damage, or leaves an ankylosis behind in one or two joints. (3) A mono-articular inflammation—either acute, and terminating more often in ankylosis; or chronic, with much effusion and little pain. (4) A form in which the bursæ and tendinous sheaths are affected, apart—it may be—from any articular inflammation. Occasionally also, great tenderness of the heel is met with, which appears to be due to a localised periostitis. Complications are sometimes met

with, such as conjunctivitis, neuritis (especially sciatic), endocarditis, and pleurisy.

As to *treatment*, careful attention should be paid to the cure of the urethral disorder. The patient should rest in bed to begin with. In order to ensure more complete rest of the affected joint, splints are often helpful. Good, nourishing food is desirable, and everything should be done which is likely to improve the general health. Medicines, apart from tonics, are of little service. Little can be done locally in the acute stage beyond fomentations and anodyne applications to relieve pain. Later on, massage, passive movements of the joint, mercurial dressings, and occasional blistering are recommended.

Change of air, a sea-voyage, or a course of Spa-treatment is strongly advised in order to complete convalescence.

RHEUMATOID ARTHRITIS.

Under this heading is included a group of arthritic affections variously described as arthritis deformans, osteo-arthritis, and rheumatic gout; which, though presenting acute and sub-acute forms, with slight or no changes in the cartilaginous and bony structures of the joints, yet have a strong tendency both to chronicity and marked joint-changes, involving more or less deformity. Its essential nature is doubtful, but evidence of an infective origin is being accumulated, though precise details are lacking.

Taking all types of the disease together, its earliest manifestations are met with most frequently between thirty and forty years of age, but there are wide variations. In general terms, the acuter forms with relatively slight changes in cartilage and bone (if any) are more common in young adults, though children and aged persons are by

no means exempt. On the other hand, chronic forms, with marked deformity, occur chiefly in later life. In some cases we find evidence of predisposing causes—namely, hereditary influence, discharging wounds, antecedent infective disease such as influenza or pyorrhœa alveolaris, prolonged mental strain or worry, or exhaustion incidental to child-bearing; but very often none can be discovered.

ANATOMICAL CHANGES.—Clinically, rheumatoid arthritis falls into two main groups. In the one, the changes are almost confined to the *peri-articular* structures; and changes in the cartilage, when present, are slight and local. In the other, bony overgrowth is the predominant feature—osteo-arthritis. It will be convenient, however, to take the various joint-changes together. *Effusion* often occurs, especially in the more acute forms. The *synovial membrane*, particularly in peri-articular cases, is swollen and injected. There is often marked proliferation of the marginal fringes, forming villous processes. Later on, the latter may become calcareous; and, when—as sometimes happens—one becomes detached, it forms a “loose body” in the joint cavity, which may be troublesome when it shifts its position. The *ligaments* and fibrous structures around the joints are apt to become thickened and sodden. The most characteristic change in the *cartilages* is erosion. In peri-articular cases it is slight, and limited to certain parts in contact with the synovial fringes. But otherwise it may be general, and proceed to the destruction of the entire cartilage, resulting either in a bony ankylosis, or the formation of a bone-surface of the smoothness and hardness of ivory (eburnation). The *bones* in many of the acuter forms show little or no change; but, in chronic cases, there is characteristic bony overgrowth. This is most marked at the outer boundaries of the joints, and often invades parts of the surrounding ligamentous or fibrous tissue. More rarely we meet with

atrophy of the ends of the long bones. Muscular atrophy is a common feature, though it varies widely in degree. It is due partly to disuse, but partly (it would seem) to trophic influences.

SYMPTOMS.—1. *Peri-articular type.* Some cases are characterised by repeated acute attacks, the patient being relatively well in the intervals between them. In a much larger proportion the affection is slow, gradually progressive, and chronic. In either the acute or chronic form the first onset may be sudden or gradual; but whereas sudden onset is more common in acute cases, most chronic cases develop gradually. Also in each form alike, several joints may be attacked simultaneously, or the process may begin in one joint, and then extend to others in succession.

The *acute* form may closely resemble an attack of acute rheumatism at the start. The onset is more often abrupt, affecting many joints simultaneously. The affected joints are painful, tender, and swollen—mainly as the result of synovial effusion. The cervical spine, temporo-maxillary and sterno-clavicular joints, which are rarely affected in acute rheumatism, may be involved. There is moderate fever, but chills and sweating are unusual. More or less enlargement of the spleen and lymphatic glands is relatively frequent. Recovery is slow and tedious, and some persistent thickening of the affected joints is generally left behind.

The *chronic* form, which is much more common, develops gradually as a rule. It is apt to begin in the fingers or knees, and only extends to other joints at considerable intervals, so that it may be a long time before it has become at all general; and its progress is frequently broken by periods of remission or intermission. As a rule its distribution is symmetrical. Pain is usually the first symptom. It varies greatly in degree, and bears no definite relation to the local changes in the joints. The

latter meanwhile become enlarged, mainly due to thickening of the peri-articular structures, but partly to synovial effusion in some cases. Movement in the joints becomes restricted, and is sometimes altogether prevented by local thickening and adhesions. Later on, contractures may take place, giving rise to more or less deformity. The deformity of the hands is very characteristic, the fingers overlapping each other, and leaning to the ulnar side, while the phalanges are variously bent. The muscles are wasted, especially in the neighbourhood of the affected joints. The deep reflexes are generally increased, and muscular cramps are of frequent occurrence. A persistently frequent pulse, moist, clammy skin, brown freckle-like spots on the skin, and ribbing of the nails are common and somewhat characteristic features. The temperature is either normal or very slightly raised; and, beyond anæmia and some liability to dyspepsia, the general health may be good. Glandular enlargements are less common than in the acute form. Small subcutaneous nodules are sometimes found about the elbows or wrists, or on a finger, freely movable, sometimes tender, and of temporary duration. The articular condition, known as *Still's disease*, occurring in children, and characterised by polyarthritis with enlargement of the spleen and lymphatic glands, is probably only a form of rheumatoid arthritis.

2. *Osteo-arthritic type*.—Here we meet with both generalised and local varieties. It is difficult at times to draw a hard-and-fast line between the generalised variety of this type and some of the later developments of certain peri-articular cases; but the former, apart from early prominence of bony overgrowth, begins later in life; the joint enlargements are more irregular and nodular; muscular atrophy is far less marked; and the deformities are not due to contractures, but to direct displacements from

bony growths. The pulse frequency is not increased, and enlargements of the spleen and glands do not occur.

Certain local varieties may be briefly noted. (a) *Heberden's nodes* are small, bony knobs, which develop at the sides of the distal phalangeal joints, generally affecting both hands symmetrically. They are painless for the most part, but sometimes both painful and tender. They may exist alone, or be associated with other manifestation of rheumatoid arthritis. (b) Mono-articular varieties are also met with, especially in old men, more commonly affecting the hip or shoulder joint. The chief features are pain, with progressive limitation of movement, culminating in immobility. Muscular wasting is well marked. (c) *Spondylitis deformans* is the term applied to rigidity of some part of the spine from bony ankylosis of the vertebræ. It is apt to cause severe pain and muscular atrophy from pressure upon the spinal nerve-roots.

TREATMENT.—Tonic medication is of most service, such as quinine, iodide or arseniate of iron, and cod-liver oil. Guaiacol carbonate, given in doses of gr. v. three times a day, and continued for a long period, has been strongly recommended. Great benefit is often derived, especially in the earlier stages, by a course of douche-massage and passive movements, as carried out at Aix-les-Bains, and adopted in many of our English health-resorts; but it should never be employed unless the joints are in a quiescent condition. The diet should be generous. Animal foods and fats may be freely taken, and a little light wine or diluted spirit is often desirable.

GOUT—PODAGRA.

Gout, though largely *hereditary*, may also be *acquired*. It is more common in men than in women; and, as a rule,

it does not occur before middle life, but with a strong hereditary taint it may appear earlier.

Among the factors which favour the occurrence of gout, none are of greater importance than the dietetic habits of the patient; and of these, pre-eminence must be given to the habit of free indulgence in the heavier wines and malt liquors. Of scarcely less importance is the habit of frequent hearty meals containing a large proportion of proteid ingredients, the bad influence of which is accentuated by association with rich carbonaceous food, and by sedentary habits. On the other hand, an active, vigorous, outdoor life will go far to discount the harmful effects of too liberal eating and drinking.

Gout is characterised by an excess of uric acid in the blood, where it is said to exist in the form of soluble quadriurate of sodium, which on decomposition results in the formation of relatively insoluble bi-urate of sodium, followed by its deposit in a crystalline form in the cartilages of the joints and other tissues. While excess of uric acid in the system is obviously favourable to deposition, some further factors are necessary in order to fully account for it, because in leucocythæmia, where there is even greater excess of uric acid than in gout, no such deposits take place. The reason of this apparent anomaly is not quite clear; but it may possibly depend upon some difference in the form in which uric acid exists in the blood of gouty subjects, which either renders it more liable to precipitation, or more difficult for the kidneys to eliminate it. Among the factors which favour uratic precipitation, the most important is the proportion of sodium salts in the medium in which it occurs. Blood, lymph, synovial fluid, cartilage, and fibrous tissue contain a proportion of sodium salts which is three to four times greater than in other parts of the body. The motion of the blood and lymph, however, hinders precipitation, while

the stationary condition of synovial fluid favours it, and not only leads to deposit of bi-urate in this fluid, but in the cartilage and fibrous structures which it bathes—assisted by the high proportion of sodium salts which these tissues contain.

Without discussing the various theories of gout which have been advanced from time to time, it seems to the writer that the evidence on the subject strongly suggests that the cardinal feature of gout is a slow and perverted metabolism, chiefly affecting the proteid constituents of the food and tissues, thereby leading to increased formation of uric acid, or some of its precursors—a metabolic defect which may be either inherited, or acquired in after life, possibly under the influence of nervous, hepatic, or digestive disturbance, or of defective elimination. Extension of analogous metabolic defect to other elementary principles, such as fats, carbohydrates, and cholesterin, might possibly explain the liability to obesity, glycosuria, and gall-stones in gouty subjects.

According to the view here expressed, excessive supplies of proteid food which overtax metabolism, excess of carbohydrates which spare proteid metabolism, alcohol which disturbs it, sedentary habits which lessen its activity, and nervous influences which pervert or depress it, are just the agents which would be most likely to favour the occurrence of gout; and such are exactly the facts resulting from clinical observation.

Gouty deposits are most common (for the reason above stated) in joint-cartilages, ligaments, tendons, and subcutaneous tissue (especially of the outer ear), and set up irritation or inflammation according to the rapidity and extent of their occurrence. The proximal joint of the great toe is generally first attacked; but, as the disease progresses, it tends to involve other joints. The deposit is crystalline, and first occurs in the substance of the

articular cartilage in the form of specks, streaks, or patches, while later on it may involve all the fibrous structures of the joint. When the deposit is subcutaneous, nodules of variable size are formed, which are called “tophi”; or, from their white chalk-like appearance, “chalk-stones.” The skin which covers these concretions may ulcerate, and discharge a thick creamy fluid, containing bi-urate crystals in abundance.

While the theory of mechanical damage meets with general acceptance as the cause of the local manifestations of regular gout, yet, since no gross uratic deposits have been seen in the structures and organs involved in irregular gout, some have assumed that the phenomena of the latter are due to a direct toxic effect of uric acid. But this is difficult to believe; and it is much more probable that irregular, like regular gout, results from physical damage by uratic precipitation, though occurring in a less obvious form.

SYMPTOMS.—When gout is limited to the joints it is said to be *regular*, and in this form is *acute* at first, but is afterwards apt to become *chronic*. When, however, it is characterised only by internal functional disturbance, it is termed *irregular*.

An attack of *acute regular gout* is generally preceded for a variable time by digestive troubles, acidity, heartburn, nervous irritability, and scanty acid urine; but it may occur suddenly without previous warning. The onset is usually during the night, in the course of which the patient awakes with violent pain in the proximal joint of one great toe. This is soon followed by heat, redness, and swelling of the skin over the joint, which then becomes acutely tender, smooth, shining, and marked by blue dilated veins. These changes are accompanied with more or less febrile disturbance, irregular sour-smelling sweats, anorexia, constipation, and extreme restlessness. The

urine is scanty, high coloured, with a thick deposit of urates, though *the total amount of uric acid is less than normal*. The symptoms usually abate somewhat during the daytime, and return each night. In the course of about a week (in early attacks) the pain, redness, and swelling of the joint subside, the skin over the joint desquamates and gradually resumes its normal condition. Sometimes the mischief is not confined to a single big toe, but also involves the opposite toe, or other joints, either simultaneously or in rapid sequence. Under these circumstances the attack lasts longer—possibly two or three weeks. When it passes off, the patient often feels wonderfully better, and he may remain free for a long time. But recurrence usually takes place sooner or later, and often exhibits a more or less periodic tendency ; but consecutive attacks tend to become less acute and of longer duration, to involve a larger number of joints, to attack other parts such as the heel or the instep, and to return at gradually shorter intervals as the case proceeds, until it finally passes into the *chronic* form. During the intervals between attacks, the daily output of uric acid in the urine is apt to be considerably below normal.

In *chronic gout* there is less fever, and the pain though less acute is more persistent. Several joints are affected, around which “chalk-stones” are apt to form, causing enlargements, deformities, and stiffness. Nodular deposits of a similar kind may occur in the lobule of the ear, and elsewhere. The health becomes progressively impaired. Thus, the complexion is sallow, while the patient complains of various dyspeptic troubles, irregular action of the bowels, and general nervous irritability. The pulse is usually of high tension ; the heart is often hypertrophied ; and the urine is abundant, of low specific gravity, and often contains a little albumen. In point of fact, the patient is apt to drift into Bright’s disease of the granular

type, with a liability to all the complications associated with that condition.

Irregular gout is the term applied to various functional disturbances met with in gouty subjects, often quite independently of any articular trouble. They may affect the *nervous system*, causing headaches, neuralgia, or cramps; the *alimentary system*, causing nausea, vomiting, digestive disorder or constipation; the *cardio-vascular system*, causing irregular or intermittent action of the heart, palpitation, cardiac or arterial sclerosis, atheroma or angina pectoris; the *respiratory system*, causing bronchitis, or asthma; the *skin*, causing general cutaneous irritation, or eczema; the *urinary system*, causing albuminuria, granular kidney, or calculus. The liability to obesity, diabetes, and gall-stones has already been alluded to.

For the purposes of *diagnosis* the following table * may be of service:—

ACUTE GOUT.	ACUTE RHEUMATISM.	RHEUMATOID ARTHRITIS.
1. Rare in early life. 2. Smaller joints chiefly affected, especially the great toe. Asymmetrical. Joint very oedematous, red, smooth, shining, with enlarged veins. Cuticle finally desquamates. 3. Bi-urate of soda deposited in the joint. 4. Attacks of short duration, and with great tendency to periodical recurrence. 5. <i>Complications.</i> — Various functional disturbances of brain, stomach, heart, or lungs. 6. <i>Urine.</i>—Scanty, acid; often deficient in uric acid. Albuminuria common.	1. Common in early life. 2. Medium-sized joints chiefly affected. Migratory. Swelling of joint mainly due to effusion within it. Skin only slightly altered. No enlargement of veins. No desquamation. 3. No deposit of bi-urate of soda. 4. Attacks of longer duration, with less liability to recur. No periodicity. 5. <i>Complications.</i> — Endocarditis, pericarditis, and inflammation of lung or pleura. 6. <i>Urine.</i>—Febrile; sometimes slight albuminuria.	1. May occur at all ages. 2. Smaller joints more often first affected, especially the fingers. Symmetrical. Considerable swelling, and thickening of all joint structures, followed by deformity. 3. No deposit of bi-urate of soda. 4. Special tendency to become chronic. 5. No special complications. 6. Nothing special in the urine.

* Adapted from Roberts' "Handbook of Medicine."

PROGNOSIS.—Gout usually runs a chronic course with little direct danger to life. Death, when it occurs, is due either to some complication or secondary disease.

TREATMENT.—Plain living, a restricted amount of animal food, avoidance of alcoholic stimulants, plenty of exercise in the open air, encouragement of the action of the skin and bowels, and as much freedom as possible from worry and anxiety will suffice to ward off, if not altogether to prevent, an attack of acute gout in the early stages of the disease. In the more chronic forms, however, the patient is often so crippled, and his general health so broken, that a liberal diet with stimulants may become necessary.

During an attack the joint must be rested, and swathed in cotton-wool. Internally, *after* the bowels have been freely opened, it is well to administer full doses of salicin in combination with bicarbonate of potassium and colchicum (vini ℥x.-xx.) three or four times a day, until the attack subsides. The salts of potash are preferable to those of soda. If there is great restlessness and sleeplessness, Dover's powder may be given at bed-time. Generally speaking the emunctories should be encouraged to act pretty freely by systematic bathing, followed by friction of the skin, drinking of mild alkaline diluents in abundance, and the occasional use of an aperient. The diet should consist of milk, diluted with barley-, potash-, or Vichy-water. In the chronic forms of gout, alkalies freely diluted, guaiacum, and iodide of potassium are of value, but attention to dietetic and hygienic matter is of still greater importance.

OBESITY.

Obesity, or excessive stoutness, is met with under various circumstances. Among its most obvious causes are an over-supply of nourishment, or deficient utilisation of

food from bodily inactivity, or both conditions combined. No doubt a tendency to obesity is often hereditary; and it is equally true that it may also be acquired, quite apart from over-eating or deficient exercise. In such cases it is probably due to a metabolic disorder, akin to that of gout and glycosuria, with which it is not uncommonly associated. There is also a well-known tendency to obesity after forty-five or fifty years of age, especially in women; who, from the cessation of menstruation and child-bearing, have probably a much reduced index of metabolic activity, and require much less food, yet, from easier circumstances of domestic life, often drift into indolent and luxurious habits, and take as much food as before, or more.

SYMPTOMS.—By adding to the weight and bulk of the body, obesity increases the strain of exertion, and encourages inactivity, the influence of which tends still further to increase obesity, and thus a vicious circle is established. It would be a mistake, however, to assume that fat people are necessarily lethargic. On the contrary, obesity is sometimes associated with intellectual activity and power of a high order. The action of the heart is apt to be embarrassed by encrustation with fat, and may thus give rise to dyspnœa on exertion, and occasionally to œdema of the legs. The arteries are also prone to premature degeneration; and sudden death may take place from syncope, apoplexy, or angina. Owing to fatty infiltration of the liver, and mechanical interference from visceral deposits of fat, gastro-intestinal disorder and constipation are frequently met with. Dragging of the heavy abdominal walls may give rise to back-ache, especially after walking or standing; and hernia is not uncommon. The skin usually acts freely, but eczema and intertrigo are apt to occur in the folds of the body-surface. Obesity involving an increase of body-weight—say, over 25 per cent. in excess of average

weight for height—is distinctly unfavourable to longevity; and justifies an extra premium in connection with life-assurance.

TREATMENT.—The chief indications are the reduction of food-supply and encouragement of metabolism. Various dietaries have been proposed; but they all agree in substantial reduction of the total in-take, and of carbohydrates in particular, especially sugar. A liberal allowance of proteids is desirable (Banting allows 6 oz. daily); but fats should be taken sparingly. All forms of alcohol are better avoided, and only a minimum of fluid should be taken *with* meals; but a glass of hot water may be sipped with advantage one hour *before* meals, and at bed-time. When obesity depends upon metabolic disorder, it may be wise to substitute milk, cheese, and vegetable proteids for meat—at least to some extent. Only three meals should be taken daily; and, when digestion is efficient, a morning and evening meal, with light refreshment midway between, is a preferable arrangement. Regular graded exercise should be encouraged; and when this cannot be carried out for any reason, systematic massage should be substituted. Regulation of the bowels is important, and a course of treatment at Harrogate, Kissingen, or Carlsbad may be very beneficial. All treatment should be cautiously carried out; and it is seldom desirable to permit reduction of weight beyond the rate of about 3 lb. weekly. Moderate obesity, without discomfort of any kind, and stationary, is best left alone.

RICKETS—RACHITIS.

Rickets is a form of *general* malnutrition, which is characterised by peculiar changes in the bones, associated with muscular weakness, lax ligaments, mucous catarrhs, and nervous over-excitability. It is essentially a disease of

childhood, and seldom occurs before the 6th or after the 18th month. It is primarily induced by food which is deficient in fat; and if also deficient in proteids and earthy salts—either or both—the disease is more severe. Anything, however, which interferes with development and nutrition, such as inherited weakness or faulty sanitation, favours its occurrence.

MORBID ANATOMY.—The changes in the long bones consist of an excessive proliferation of the deeper layers of the cartilages and periosteum, together with delay in the process of ossification. They are consequently increased in thickness, especially at their ends; and, moreover, they become softened, so that they are peculiarly apt to yield under pressure, and give rise to deformities. The cranial bones remain membranous at their margins; the inner tables often atrophy, and bosses form on the central areas of the outer tables. After a while ossification is once more resumed, and the new-formed bone is extremely hard and dense. The liver, spleen, and lymphatic glands are also sometimes enlarged.

SYMPTOMS.—Three signs stand out as particularly characteristic of the onset of rickets:—(1) Profuse sweating of the head, face, and upper part of the chest, increased during sleep, or on the slightest exertion when awake; (2) a strong desire to be cool at night by kicking off the bed-clothes; and (3) a peculiar tenderness of the body when moved or pressed, which indicates the beginning of morbid changes in the bones (Jenner). Notwithstanding that rachitic children are sometimes unusually fat, the flesh is soft and flabby, the skin is pale, the bowels act irregularly, the motions are offensive, and the appetite is often ravenous. The cutting of the teeth is delayed, and if the child has already begun to walk, it now ceases to do so, sits “all in a heap,” suggesting to an anxious mother that the back is “growing out.” The child is weak, languid, peevish, and restless.

Before long the ends of the long bones become enlarged. This is most easily observed at the junction of the ribs with their cartilages, but enlargement of the wrists, ankles, and other joints is also very commonly observed. The bones also become softer and more or less thickened throughout. If the disease is unchecked, deformities follow. Thus, the limbs may become bowed, the ribs may be drawn in anteriorly by the action of the diaphragm, thrusting the sternum forward (*pigeon breast*) ; the spine may be variously curved ; and, in extreme cases, the antero-posterior diameter of the pelvis may be shortened by pressure of the sacrum forwards into the pelvic cavity. The head is large, out of all proportion to the face ; the forehead is high, square, and projecting ; and the closure of the fontanelles is delayed. Owing to the resistance offered by the solid organs in the upper part of the abdomen, the lower ribs are not drawn in like those above, and this gives rise to a relative enlargement of the lower part of the chest. The abdomen is large and distended.

As the disease progresses, the child becomes shrunk and old-looking, the digestive functions are increasingly disturbed, the bowels are most irregular, and the nervous system acquires a peculiar excitability and liability to convulsive disturbance from slight causes. The urine is increased in amount, and often turbid from earthy phosphates. Catarrhal and nervous disorders are common enough, and often prove fatal, but as a rule the affection is amenable to treatment if not too long delayed. During convalescence, ossification of the softened bones takes place, so that they once more become hard and firm ; but stunted growth, and persistence of any deformity which may have already taken place, constitute permanent indications of the past disease.

Of intercurrent disorders, bronchial catarrh, diarrhœa, laryngismus stridulus, convulsive affections generally, and

chronic hydrocephalus, are most commonly met with. Although in a certain sense they may be spoken of as *complications*, yet it should be remembered that they stand in the same relation to the general morbid condition as the changes in the bones.

The *diagnosis* in well-marked cases is attended with no difficulty whatever. In the milder forms, most reliance may be placed on the initial symptoms (as already described), the delayed dentition, and the lateness in walking and talking. It is distinguished from *mollities ossium* in that the latter is a disease of adults, and is never followed by re-ossification.

TREATMENT.—The diet claims foremost attention in the treatment of rickets, and many cases may be cured entirely by qualitative adjustment of the food. Fresh cows' milk diluted with barley-water according to the child's age and digestive capacity, with added proteid and fat in the shape of fresh meat-juice and cream, is the food which is most generally suitable. The addition of citrate of soda (gr. ij. to the ounce) increases the digestibility of cow's milk; and if it still disagrees it may be pancreatised. Unmalted starchy food should be altogether avoided by rachitic infants under one year old. With older children, the milk diet may be further supplemented by animal broths and the yolk of egg. Digestive disorder may call for modification of the above diet; but the principle of providing a sufficiency of proteid and fatty food must not be lost sight of. Abundance of fresh air, sunlight, and cleanliness of surroundings rank only next in importance to diet. Drugs are useful adjuncts in many cases, especially cod-liver oil, malt extract, and preparations of iron. Lime salts have also been recommended; and the author has often seen benefit from a course of chloride of calcium (dose, gr. i.-iiij.). Where the bowels are irregular and the stools offensive, a mild aperient is necessary at first, followed by a few grains

of some alkaline bicarbonate, in combination with nuxvomica or some vegetable bitter, which may be repeated three or four times a day for a while. Obstinate constipation is best treated by warm clothing, frictions of the abdomen, and occasional enemata; should any aperient be necessary, a dose of castor oil may be given; or better still, one or two drops on sugar, once or twice a day, of a solution of podophyllin in spirits of wine (gr. j. ad 3j.). On the other hand, if there is diarrhœa, the diet should be limited for a few days to albumen water or diluted meat-juice, with full doses of bismuth subnitrate (with or without the addition of $\frac{1}{4}$ to $\frac{1}{2}$ a drop of tr. opii). As to the deformities, children should be prevented from getting about too early; and in recent cases, where the bending of the limbs is marked, gentle extension with splints may be employed. Intercurrent disorders must be treated as they arise.

SCORBUTUS—SCURVY.

Scurvy is a disorder which primarily depends on a deficiency of *fresh* food. "Freshness," in this sense, is something more than mere absence of ordinary decomposition. It is a quality attaching to living matter (or that which has recently lived), whether animal or vegetable, which is destroyed by sterilisation, which is essential to proper nutrition of the body, and cannot be supplied by any manufactured food. No scientific definition of freshness is available; and, so long as this remains so, scurvy cannot be scientifically explained. As powerful auxiliary causes, cold, damp, hardship, and depressing influences generally, must be mentioned.

MORBID ANATOMY.—The blood is profoundly altered, being liquid and dark coloured. Its alkalinity is also much

reduced (Wright). Ecchymoses are seen on the surface of the skin, serous and mucous membranes. Firm coagulated extravasations are also met with beneath the skin, and between the muscles. Blood-stained effusions may occupy the serous cavities.

SYMPTOMS AND COURSE.—A sense of lassitude, debility, and disinclination for exertion of any kind, often accompanied with darting pains in the limbs and joints, usually precedes the characteristic changes of the gums, which become swollen, soft, and spongy, and bleed on the slightest touch. The teeth are tender and loosened. Subcutaneous hæmorrhages (*petechiæ*) occur, and are most abundant over the lower limbs; while not infrequently deep-seated extravasations give rise to brawny indurations about the hams and elsewhere, rendering all movements of the limbs difficult and painful. Hæmorrhages from the various mucous surfaces may occur in severe cases. The general condition is characterised by debility and prostration, the skin is sallow and harsh, the breath is offensive, the appetite fails, and the bowels are generally constipated. There is no fever in uncomplicated cases. If not relieved, the prostration increases, the pulse becomes very feeble, the least exertion is attended with dyspnœa and palpitation, and the integument is often œdematous and sometimes the seat of obstinate ulceration. Prompt and appropriate treatment is usually followed by recovery; otherwise the case drags on from bad to worse, till death occurs from exhaustion or some complication.

TREATMENT.—It is important without delay to give fresh food, to the want of which the disease is primarily due. For this purpose, milk, eggs, fresh meat-juice, pulped fresh vegetables, and fruit should be freely supplied. Lime or lemon juice (3i.-3ij. in a tumblerful of water, and sweetened, taken thrice daily) is also of great value.

The diet should be light, nutritious, and liquid until the

condition of the gums has improved, and the digestion is stronger. Stimulants are occasionally required.

Beyond simple antiseptic mouth-washes, and medicines for regulation of the bowels, drugs are not specially needed.

INFANTILE SCURVY.

A form of scurvy is sometimes met with in infants, which, from its occasional association with rickets, and from the resemblance of some of its symptoms to those of rickets, has been mistaken for that disease, and called "acute, or scurvy rickets." But it is a true scurvy; and results from exclusive feeding on manufactured foods, condensed milk, or milk submitted to prolonged sterilisation—in short, any food deficient in the element of freshness.

The disease develops rather quickly, the child becoming pale, and very fretful, crying whenever moved or pressed, owing to tenderness of the bones. Examination of the latter reveals ill-defined and acutely tender swellings—due to sub-periosteal hæmorrhage—on the shafts of the long bones in the neighbourhood of the epiphyses (more often affecting the legs than the arms), on the ribs, or more rarely on the bones of the cranium. One also meets occasionally with protrusion of the eyeball from similar hæmorrhage in the orbit; and separation of epiphyses may occur, giving rise to crepitus on movement. Before dentition, the gums are unaffected; but otherwise they are swollen, spongy, and readily bleed. The skin bruises on very slight provocation, sub-conjunctival hæmorrhage is not uncommon, but purpuric spots on the skin seldom occur. In severe cases hæmorrhages from mucous surfaces may be observed. The temperature is very erratic—in some cases normal, in others moderately febrile. The general condition is characterised by anæmia and debility, but the appetite is usually fair, digestion is

but little disturbed, and there is no emaciation. The urine frequently contains a little albumen.

The characteristic lesions consist of sub-periosteal hæmorrhages, occurring without any inflammatory manifestations, and causing more or less absorption of adjacent bony structures. They do not suppurate.

The essential feature of treatment consists in supplying fresh food, in the form of fresh milk, sieved potato, fresh meat-juice, and a tablespoonful (in divided doses) of orange or grape-juice daily. With early treatment recovery is the rule. No infant should be fed on manufactured foods exclusively, at any rate after six months of age.

PURPURA.

Purpura is characterised by subcutaneous hæmorrhages, which vary in size from minute petechiæ to large ecchymoses, and present those changes of colour with which we are familiar in bruises. Next to nothing is known with regard to its pathology, but there is probably some disorganisation both of the blood and of the walls of the small blood-vessels.

In many cases purpura is obviously *symptomatic*. Thus it may be met with in specific fevers, giving rise to hæmorrhagic varieties; in profound cachexia—from scurvy, cancer, senile decay, and the like; in toxic conditions—from snake bites, or certain medicines; and as an occasional result of irritative nervous lesions.

Primary purpura, in a mild form (*purpura simplex*), is a common affection among children. The purpuric spots are most abundant on the legs and lower part of the trunk, and frequently appear in successive crops. There may be fleeting joint-pains, but there is little or no fever or general disturbance, and recovery occurs within a fortnight. In a severer form (*purpura hæmorrhagica*), most

common in delicate girls, the spots are associated with profuse hæmorrhages from the mucous membranes of the nose, alimentary canal, kidneys, or air-passages. Profound anæmia follows, with great prostration. Recovery may take place as in the milder form, after a slow convalescence ; but it is not infrequently fatal.

There is a third variety of purpura, known as *peliosis rheumatica*, in which the spots are associated with moderate inflammation of the joints and pyrexia. The spots are mainly confined to the lower limbs, especially the front of the legs, and are slightly raised (*purpura urticans*). There is little constitutional disturbance. Recurrence is common. The affection is most frequent in males between the ages of twenty and thirty years.

Scurvy is distinguished from purpura by the marked cachexia, affection of the gums, and dependence upon a want of fresh food. Hæmorrhages from mucous surfaces are also more common and abundant in purpura than in scurvy.

The *treatment* is necessarily of a general kind ; and attention to the hygienic surroundings of the patient, a liberal supply of good food, with warmth and rest, should constitute its leading features. Medicinally, iron and quinine with sulphuric acid are of most service. Internal hæmorrhages are best checked by the administration of turpentine in doses of ʒ x.-xx., which may be compounded with *mistura amygdalæ*.

HÆMOPHILIA.

Hæmophilia is a constitutional and hereditary tendency to profuse bleeding, either spontaneously, or (more commonly) after some traumatic injury. The affection is more common among males than females ; but it may be transmitted by the latter to their children, though they

themselves do not bleed. Nothing is known with regard to its pathology, but it is closely related to a serious diminution in the coagulability of the blood.

Apart from wounds, the most frequent bleeding is from the nose, and next from the gastro-intestinal tract. Hæmorrhages into the joints are not uncommon, and may give rise to some arthritis. Legg has divided the disease into three degrees, according to the severity of the symptoms: (1) Internal and external bleedings of every kind, together with joint-affection; (2) Internal bleedings only; and (3) Ecchymoses only. Death is common before eight years of age, from loss of blood; but the longer a patient survives beyond this age the better the prognosis.

Beyond protecting the members of a bleeder's family from injury, and forbidding the daughters from marrying, little can be done.

DIABETES MELLITUS.

Diabetes is characterised by the persistent presence in the urine of grape-sugar (glucose) in more or less abundance, attended with constitutional disturbance of a marked kind. Normal urine is said to contain a very minute trace of sugar, and we meet with temporary glycosuria in various morbid conditions, but such are not included in the following description.

Diabetes is undoubtedly hereditary in some cases, while in others it may be associated with obesity or gout, or it may follow parturition, or a specific fever. It is much more frequent at and after middle life than in the young, and in the male than in the female. Exposure to cold, violent emotion, nervous strain and worry, are among the chief exciting causes, but very often none can be traced.

The pathology of diabetes is very obscure, and the

present state of our knowledge admits of little more than speculative probabilities. The various theories cannot be discussed here in detail, and it must suffice to indicate in general terms some of the more important points. There is no doubt that the presence of sugar in the urine in appreciable amount corresponds to an excess of sugar in the blood. How is this excess to be accounted for? Only two alternative answers are possible—increased supply or diminished utilisation of sugar. In the absence of all evidence of the former, it is to the latter that we must turn for reply. All carbohydrates and sugars are passed into the portal blood in the form of glucose, the majority of which is taken up and utilised by the liver, being partly deposited in the form of glycogen. The remainder probably passes by into the general circulation to be utilised by the tissues, by a process of splitting up, coupled with oxidation. Thus it will be seen that there are two points at which utilisation of sugar may fail—the liver and the tissues. The simplest form of hepatic failure is afforded by the glycosuria which follows certain nervous lesions, which has been almost conclusively proved to be attended with the disappearance of glycogen from the hepatic cells, suggesting that, under these circumstances, sugar cannot be adequately held up by the liver, and thus passes on into the general circulation.

The conditions under which the tissues of the body fail to metabolise sugar are very imperfectly understood. In a certain number of cases it appears to be closely related to disease of the pancreas, and there is strong probability that this gland yields an internal secretion quite distinct from pancreatic juice, which in some way is necessary to the cleavage and oxidation of the sugar molecule. There is no proof, however, that this relation applies to all cases of diabetes. Some cases, with great probability, may be referred to nervous influences like that which disturbs the

glycogenic function of the liver ; and yet again there may be cases which have an intestinal origin connected with the absorption of toxic substances from the digestive tract.

In mild cases of diabetes the amount of unconsumed sugar is small ; but when severe, not only does the body fail to consume the carbohydrates supplied in the food, but also the carbohydrate moiety of proteid metabolism. In all but the mildest cases, diabetes is accompanied by the production of considerable quantities of organic acids, chiefly oxy-butyric and diacetic, which are excreted in the urine in combination with ammonia and other alkaline bases. Loss of weight and strength result from the failure to utilise sugar ; and certain nervous symptoms, associated with a peculiar form of coma, are due to the serious withdrawal of alkali from the body in combination with organic acids (acid intoxication).

The polyuria and thirst of diabetes depend upon osmotic disturbance caused by the presence of sugar in the blood. Sugar, in proportion to its amount, determines a strong flow of fluid into the blood, which passes off into the urine, and carries the sugar with it. When this demand for fluid is not satisfied by drink, water is abstracted from the tissues and causes thirst.

Apart from a few cases of medullary tumour, and a larger number in which changes have been found in the pancreas, it cannot be said that the *anatomical appearances* in diabetes are characteristic or have any clear connection with the cause of the disease. Many of the minor lesions which have been described by Saundby and others are probably of secondary origin.

The *clinical history* of diabetes varies greatly in different cases. In children and young adults it generally takes an acute form, and ends fatally before long ; but in those past middle life the symptoms are much milder as a rule, and run a very chronic or intermittent course. But

diabetes is never free from danger, and adds an element of gravity to any intercurrent disease, especially when the latter happens to be of an acute nature.

The onset is generally insidious, and attention may be first aroused by gradually increasing weakness, thirst, more frequent passage of urine, or a crop of boils. But in whatever way it begins, the essential features of the established disease, when well-marked, are thirst, voracious appetite, progressive emaciation and loss of strength, dryness of the skin, accompanied with glycosuria.

The quantity of urine is largely increased, and varies from 5 to 12 pints daily, or even more in some cases. It is clear, with a remarkably brilliant aspect. The specific gravity is high, varying from 1·020 to 1·060, but 1·030 to 1·040 is a common average. It contains a large but variable amount of sugar, usually from about 3 to 6 per cent.* The excretion of urea is increased; moderate albuminuria is common; and in severe cases, oxy-butyric acid, diacetic acid, and acetone are also present.

The frequency of micturition is increased. Thirst and appetite are excessive; emaciation is progressive. The skin is dry, harsh, and irritable; and, not infrequently, the ankles are oedematous. The tongue is red and dry; the teeth are often loosened and are apt to decay rapidly; and the gums may recede and become unhealthy. Other less characteristic symptoms are frequently observed, such as palpitation with shortness of breath on exertion, digestive disturbance, obstinate constipation, mental dullness, volitional weakness, and weakness or absence of sexual desire.

In the milder forms of diabetes, often met with in later life, and (not infrequently) in connection with a gouty habit, or marked corpulence, all the above signs and

* The tests for sugar will be described in the chapter on "Urine."

symptoms are less marked ; thirst is not conspicuous ; the appetite may be quite moderate ; and loss of weight is very gradual. In a certain number of cases, beyond the presence of a considerable amount of sugar in the urine, there may be little or no indication of disease.

The chief *complications* of diabetes are the following : eczematous affection of the genitals, boils and carbuncles, cataract, and sciatica. Pulmonary tuberculosis is of especial importance, and supervenes in about half of all cases. Another common and fatal complication is what is known as "diabetic coma." Its occurrence appears to be favoured by over-exertion, mental anxiety, shock, or too abrupt restriction of the diet. It develops gradually with pain in the abdomen, languor, and lassitude ; respiration becomes slow and sighing ; the breath has often an ethereal smell ; the urine may be diminished in amount ; coma gradually develops, and the patient dies in from one to three days. Diabetic coma is probably related to the so-called acid intoxication above referred to. It should be added that this type of coma is not peculiar to diabetes, but sometimes occurs in cancer of the liver and other diseases.

TREATMENT.—This may be discussed under three heads : general, dietetic, medicinal.

General.—Exercise (always stopping short of fatigue), fresh air, warm clothing, and occasional baths are of considerable benefit. The bowels should be carefully regulated by castor oil, or saline aperients. The digestive functions require attention, and for this purpose nothing is better than alkalies combined with some vegetable bitter. To relieve the intense craving for food, Sir Wm. Roberts recommended a pill containing two or three grains of asafoetida twice or thrice daily. Sleeplessness may be combated with opium, bromide of potassium, and the like.

Dietetic.—The chief indication is to cut down the amount

of sugar or sugar-forming substances in the food. A good deal of judgment is required in carrying this out, for diabetic patients differ very much in their toleration of rigid restrictions, and in their ability to digest manufactured carbohydrate substitutes. Few can tolerate absolute withdrawal of carbohydrates. Nor indeed is it necessary, for the maintenance of nutrition is important, and this often necessitates the allowance of a certain quantity of bread, or (better still) well-cooked potato. In fixing the amount of carbohydrates allowable, one must be guided by the effect on the urine. A little alcohol in the form of dilated spirit is often desirable. The diet should be selected mainly from the following :—milk, butter, cheese, eggs, meat, fish, and green vegetables. Gluten bread, almond and cocoa-nut cakes, and similar preparations may sometimes be substituted for bread with advantage. Imperial drink is of service in relieving the extreme thirst.

Medicinally, opium appears to be the most valuable drug, and should be given in small doses (gr. $\frac{1}{2}$ *ter die*) at first gradually increased. Others prefer codeia to opium, which is given in similar doses. Opium should be used with caution in gouty subjects. In such cases great benefit may be occasionally derived from the administration of salicylate of soda ($\mathfrak{A}$ j. *ter die*) in combination with alkalies. With marked anæmia and emaciation, iron and cod-liver oil may be useful. Pruritus vulvæ is often a very distressing complication. It is best treated by strict cleanliness, hot effusions to the part, followed by application of a solution of corrosive sublimate (1 in 2,000), carefully dried, and then dusted with lycopodium powder. The best treatment for diabetic coma is by subcutaneous injection of a normal saline fluid, with full doses of bicarbonate of soda by the mouth ; but, at best, the chances of success are small.

DIABETES INSIPIDUS.

Diabetes insipidus is a clinical term applied to excessive and persistent increase in the urinary secretion, the urine being of low specific gravity, and containing neither sugar nor albumen (or only occasional traces). Different groups of cases have been distinguished, according to whether the polyuria is associated or not with serious increase of the solid constituents as well. In the common form, which may be taken as the type of the disease, the excretion of solid constituents is not seriously disturbed. The name "*hydruria*" has been applied by some writers to this condition.

It mainly occurs in early childhood, or middle-adult life, and is much more common in males than females. Sometimes it develops acutely under the influence of nervous injury or disease, or sudden shocks. More often it develops slowly, and is predisposed to by anything which tends to nervous weakness and depression, such as hygienic neglect, inherited constitutional disease, or prolonged worry or anxiety.

The *urine* is pale, with a sp. gr. of about 1.003-6 as a rule, and a neutral or faintly acid reaction. Although relatively much diminished, the solid constituents are increased, but not to a very great extent. The average daily quantity of urine varies from 5 to 15 pints. There is intense *thirst*. The appetite is good, and often voracious, so long as the digestive machinery holds out, but afterwards it steadily fails; the tongue becomes sticky; the bowels irregular; and emaciation sets in. As the tissues become dehydrated, the patient complains of aching pains in the back and legs. Headache is frequent; with general nervous weakness, and impotence (in male adults). The temperature is subnormal.

The progress varies. In certain cases, especially those

of the acuter type, much may often be accomplished by early treatment. In chronic types, much depends on the degree of polyuria, and the state of health prior to attack. But, on the whole, diabetes insipidus is a serious disease, frequently leading to fatal exhaustion or coma, or death from some intercurrent disease. When once digestion has broken down, life is seldom prolonged for over six months. It must not be forgotten that the kidneys are liable to break down from prolonged physiological overstrain; and for this reason death sometimes occurs unexpectedly from uræmia.

Treatment is most unsatisfactory. Ample rest, freedom from worries (so far as possible), change of scene, good food, together with nervine tonics, such as arsenic and strychnia, afford the best means of prolonging life under the circumstances. Remarkable improvement has been observed in certain cases by preparations of valerian and ergot.

SECTION IV.

DISEASES OF THE BLOOD AND LYMPH-GLANDS.

GENERAL CHARACTERS OF THE BLOOD.

THE fundamental purpose of the blood is to supply the body with nutriment, and to carry away its waste products. No doubt the fulfilment of this purpose is largely influenced and determined by physical conditions, yet it must be borne in mind that there are also important factors of a biological order relating to the blood itself, to the tissues through which it circulates, and to the vascular walls interposed between the blood and the tissues, which make the situation exceedingly complex, and about which very little is known.

The total amount of blood has been stated to be about $\frac{1}{13}$ of the body-weight; but, apart from hæmorrhage, variations in this respect are not of much clinical importance. On the other hand important clinical considerations may arise from variations in the relative amount of blood supplied to a part, under the influence of physical or vaso-motor disturbance. The specific gravity of the blood is about 1.056; which may be increased, as in diabetes, or lowered, as in anæmia. The reaction is invariably alkaline, though it exhibits appreciable variations in degree, and the marked reduction of alkalinity in diabetic coma is a striking example.

The number of *red cells* in a cubic millimetre of normal blood is about 5,000,000. This number is reduced in all forms of anæmia, but to an extent which varies widely

according to circumstances. For trustworthy calculation, a special instrument (hæmocytometer) is necessary. The average diameter of each cell is $7.5\ \mu$. A few of larger and smaller size may be met with in normal blood, but much greater variations occur in disease, especially in anæmias of severe type. Marked irregularities in shape (poikilocytosis) are also observed in disease, especially in pernicious anæmia. Nucleated red cells are not met with in normal blood, except for a short time after birth; but they are found in the graver forms of anæmia, and are known as normoblasts or megaloblasts according to their size.

The amount of colouring matter (hæmoglobin) varies independently of the number of red cells; and the relation of the percentage of the one to that of the other is called the *colour-index*. Thus, with a normal number of cells, and only 50 per cent. of hæmoglobin, the colour-index is $\frac{1}{2}$; whereas, if the percentage of cells be reduced to 25, and of hæmoglobin only to 50, the colour-index would be 2.

The number of *white cells* or *leucocytes* in a cubic millimetre of normal blood varies from about 6,000 to 10,000 per cubic millimetre. Several forms are distinguishable by their size, the character of their nuclei, and their reaction to staining fluids. In healthy blood, about 65–75 per cent. have an average diameter of $10\ \mu$, are finely granular, with subdivided nuclei, actively amœboid, and phagocytic (polymorphonuclear cells). Another 20–30 per cent. are small hyaline cells (lymphocytes), about the size of red cells, with a single nucleus, and are neither amœboid nor phagocytic. A few hyaline cells of larger size are also seen. Lastly, about 2 per cent. are large coarsely granular polymorphonuclear cells about $12\ \mu$ in diameter (eosinophiles), which are also amœboid.

An increase in the proportion of leucocytes, moderate in amount (for the most part between 15,000 and 30,000 per cubic mm.) and not associated with enlargement of the

spleen or lymphatic glands, is known as *leucocytosis*. It is met with physiologically after violent exercise, in pregnancy, and after meals. It also occurs pathologically, as a result of severe hæmorrhage, suppurative inflammation, many infective disorders, and malignant disease (especially sarcoma). The increase mainly affects the polymorphonuclear cells. It is worth noting that in typhoid fever, influenza, measles, and most forms of tuberculosis, leucocytosis is usually absent. A persistent and very great excess of leucocytes, associated with enlargement of the spleen or lymph-glands, is called *leukæmia*, and is always pathological. A deficiency of leucocytes is known as *leucopenia*.

Blood-platelets are small ovoid or circular bodies, finely granular, adhesive in behaviour, and very delicate and unstable. They are found in considerable but variable number in normal blood.

Blood-formation.—In the adult, the red-cells of the blood, and all leucocytes with granular protoplasm, appear to be exclusively formed in the bone-marrow; whereas lymphocytes are formed in the spleen, lymph-glands, and lymphatic structures of the body generally. During foetal life, such strict differentiation is not observed; both red and white cells are formed not only in all the above-named parts, but in the liver as well. The earliest stages of the *red cells* are represented in the marrow by nucleated cells, larger than those seen in the blood, which gradually acquire red colouring matter. Before discharge into the blood, these cells shed their nuclei and become smaller. Similarly, large mononuclear granular cells (myelocytes) are the antecedents of the *polynuclear leucocytes* of the blood, with an intervening stage in which the single nucleus becomes polymorphous. In certain diseases (as we shall see) nucleated red cells or myelocytes may be found in the circulating blood; and when this is so, it

should be regarded as due to a hurried discharge of half-finished red or white cells into the blood, probably as the result of a special demand for fresh supplies. The formation of *lymphocytes* in the spleen and lymphatic structures proceeds on strictly analogous lines. In the glands it takes place in groups of cells undergoing active division (germ-centres). The cells work out from the centre towards the periphery, gradually becoming smaller, until they are finally extended into the lymph stream, and so on to the blood, as fully formed lymphocytes. The behaviour of the malpighian bodies in the spleen is exactly parallel.

In the foetus, there is little difference between the structure of the marrow and lymph-glands, each turning out much the same kind of cells. Similarly, we sometimes find, in leukæmia and certain other forms of anæmia, the same approximation of structural type, and reversion to primitive function.

ANÆMIA.

The term anæmia denotes, in general terms, a depreciation in the physiological value of the blood, caused either by loss, destruction, or defective formation of red blood-cells; or by deficiency of hæmoglobin. No doubt there is generally a corresponding depreciation of the plasma as well; though, in actual practice, there is difficulty in estimating it. A useful, though somewhat arbitrary, division of anæmia may be made into a secondary or symptomatic form, and a primary or essential form including chlorosis and pernicious anæmia.

The following *symptoms* are common to all forms of well-marked anæmia:—pallor and general weakness, languor and lassitude, and disinclination for physical or mental

exertion ; loss of appetite, frequent discomfort after food, with flatulence and a tendency to constipation ; shortness of breath and palpitation on exertion, a persistent increase in the frequency of the pulse, and occasionally anasarca ; frequent headaches, bad memory, and sometimes spots before the eyes (*muscæ volitantes*), singing noises in the ears (*tinnitus*), and a liability to neuralgic pains. The muscles are soft and flabby, the extremities are chilly, the hair is apt to fall off, and the nails are often thin and ill-formed. Cardio-vascular murmurs are of common occurrence, especially in chlorosis. A systolic murmur is often heard over the pulmonary artery, in the left second intercostal space ; and a continuous venous hum (*bruit de diable*) over the large veins just above the right clavicle may be also present. These so-called hæmic murmurs result partly from alteration of the blood, and partly from the physical condition of the parts in which they are produced. A systolic murmur is sometimes heard at the apex of the heart, due to atonic dilatation with relative mitral regurgitation.

SECONDARY OR SYMPTOMATIC ANÆMIA.

The following are the most important causes of secondary anæmia. (1) *Hæmorrhage*, from wounds, ulceration, bursting of an aneurism, morbid blood-states, and parturition. If the patient survives, the number of red cells is reduced, and is associated with a leucocytosis lasting from a few hours to several days, in proportion to the loss of blood. The plasma quickly recovers its saline and watery constituents, but it takes a longer time to regain the normal proportion of blood-cells, and still longer to acquire that of hæmoglobin. (2) *Malnutrition*, from deficient supply of food, interference with digestion or absorption of food, wasting diseases like cancer, or want of exercise and

fresh air. (3) *Undue drain upon the essential ingredients of the blood*, as by long-continued suppuration, persistent discharges, over-lactation, and chronic albuminuria. (4) *Direct destruction of the red cells of the blood (hæmolysis)*, such as occurs, for instance, in malaria, blackwater fever, and acute rheumatism. (5) *Obscure interference with blood-formation*, occurring for the most part under toxic influences, as from poisons introduced from without, such as lead or mercury, or from auto-intoxications of various kinds. In actual experience several of the foregoing conditions may be in operation simultaneously.

CHLOROSIS.

Chlorosis is a disease confined to women. It generally begins between puberty and twenty years of age, but recurrence at a later age is very common. It has probably some relation to the important developmental changes which are taking place at the time when chlorosis usually occurs, but its nature is quite unknown. There appears to be a close connection, in many cases, with physical exhaustion from over-exertion, especially in in-door occupations, and among those who have exchanged country for town life. In other cases, there may be an element of auto-intoxication arising, for instance, from persistent constipation. Menstruation (if it has already appeared) usually ceases, or the flow becomes scanty; but in all probability this is merely a secondary effect of the disease.

The blood-changes are characterised by a marked reduction of specific gravity, together with a striking deficiency of hæmoglobin. As a rule, there is only a moderate reduction in the number of red cells; but in severe cases they are much reduced in number, and sometimes exhibit poikilocytosis. The white cells are not materially affected.

The *symptoms* are those common to all forms of anæmia,

and indicative rather of functional disturbance than of malnutrition. The pallor is intense, with a slightly greenish tinge, which is characteristic. The onset is gradual, and the earliest manifestations of the complaint are weakness, shortness of breath on exertion, headache, and indigestion. The latter is generally of a hyperacid type, which is curious in face of the fact that chlorotic patients are often particularly fond of vinegar and pickles. A liability to ulcer of the stomach should also be noted—a condition which is commonly associated with over-acidity. The bowels are usually constipated. The body does not waste; but, on the contrary, there is a general tendency to plumpness. Slight pyrexia is not uncommon, and a small amount of albumen in the urine is frequently present.

PERNICIOUS ANÆMIA.

This term is applied to a progressive, but often paroxysmal type of anæmia, characterised by hæmolysis, associated with special morphological changes in the blood and marrow, tending to a fatal issue, and not traceable to any known cause. Nervous shock, prostration, and anxiety appear to act as predisposing causes, and the disease is very frequently observed in connection with gastro-intestinal disturbance. The disease is evidently due to some profound toxic condition, which is responsible not only for the destruction of blood-cells, but also for the notable tissue-degeneration which is associated with it. In its *typical* form, it is most common in men over thirty-five years of age. There are other cases of anæmia which resemble true pernicious anæmia in their toxic origin, hæmolytic effects, and progress to a fatal issue; but it is said that the changes in the blood and marrow are different. Many of them are connected with pregnancy or parturition. If these be included, the age incidence is earlier, and the proportion of women considerably higher.

Anatomical changes.—Fatty degeneration is a prominent feature, especially affecting the heart, liver, and kidneys. Degenerative changes in the spinal cord are not infrequent. The bone-marrow is softened, and of a bright red colour throughout, as the result of an overgrowth of the elements concerned in the production of red blood-cells, with unusual abundance of megaloblasts. There is a remarkable deposit of iron in the cells of the liver, kidneys, and spleen, probably derived from wholesale destruction of red blood-cells. The colour of the blood (to the naked eye) is little affected owing to the high colour-index; but if a drop be allowed to fall on white blotting-paper, the central red spot is surrounded by a zone of paler colour, due to staining of the blood-plasma. The number of red cells is extremely reduced, and they vary much in size and are irregular in shape, with a certain proportion of nucleated forms among which megaloblasts predominate. The white cells are also diminished, with a relative increase of lymphocytes. The colour-index is high (rarely less than 1), owing to the large increase in the hæmoglobin content per corpuscle.

The onset is very slow and insidious. Progressive general weakness is more often the earliest symptom; but attention may be first attracted by nausea and vomiting, or by diarrhœa; while in others again, short breath and palpitation are chiefly complained of. It is remarkable, however, for what a long time in many cases nutrition and general capacity are maintained, notwithstanding the progressive pallor, which is of a canary-yellow colour—sometimes so marked as to simulate jaundice.

When well established, the chief symptoms are extreme prostration, gastro-intestinal disturbance (especially diarrhœa), headache, dyspnœa, and palpitations. Soreness of the mouth is not uncommon; hæmorrhage from mucous membranes or in the skin and retina are apt to occur; there are periods of pyrexia of moderate duration; and

the urine is pale (probably as the result of renal inadequacy from fatty degeneration), and may contain a little albumen.

In the great majority of cases, the progress is interrupted by one or more remissions, lasting from one to twelve months, in which the health greatly improves. Actual recovery does sometimes take place; but it is rare, and fatal termination is the rule.

TREATMENT OF ANÆMIA.—In secondary anæmia, the main object should be to ascertain the cause, and to remove it if possible. Fresh air, out-door exercise, plenty of sleep, and tepid or cold bathing greatly aid recovery. The food should be light and nutritious, and a little light wine or sound ale is often of service, when taken with the principal meal. Iron in some form or other is the most important medicinal remedy, and may often be usefully combined with strychnia. The tendency to constipation often calls for the addition of some aperient to the iron, of which aloes is the best. A convenient preparation is pil. aloes cum ferro, or a combination of the sulphates of iron and magnesia. Iron in any form is apt to disagree where the tongue is foul and the stomach disturbed. In this case it is better to begin with a purgative, followed by a short course of an alkali (or mineral acid, as may be indicated) combined with some vegetable bitter. Among other remedies may be mentioned arsenic and phosphate of lime. The former is of great service in anæmia arising from hæmorrhage; and the latter was recommended by Dr. Ringer for the anæmia of rapidly growing children, or women weakened by suckling.

Chlorosis is very amenable to treatment by iron, and the dose is of more importance than the form of preparation. A very satisfactory form is a pill of gr. v. of dried sulphate of iron, made up with glycerine of tragacanth, to be taken thrice daily after meals. Constipation needs strict attention. Rest for at least one hour twice a day is an important

factor of success ; and, in bad cases, confinement to bed for a time may be necessary.

In *pernicious anæmia*, iron is of no use, but a steady course of arsenic (in increasing doses) is often of the greatest benefit, and preparations of red bone-marrow may be useful. Intestinal antiseptics, such as salicylate of bismuth, and guaiacol carbonate are worthy of trial. Rest, in the completest sense, is even more urgently indicated than in chlorosis.

LEUCOCYTHÆMIA—LEUKÆMIA.

In leukæmia there is a persistent increase in the leucocytes of the blood, which is associated with changes in the spleen, lymphatic glands, or bone-marrow. No hard-and-fast distinctions of type according to distribution of lesions can be maintained. All that can be said is that the morbid process sometimes begins and preponderates in one part of the blood-making organs and tissues, sometimes in another. The rate of progress also varies, some cases running a rapid and acute course, while others (forming a large majority) are chronic. The longer the disease continues, the greater the tendency to involve *all* parts of the hæmopoietic system. Notwithstanding this close relationship in distribution, we may recognise two different types, which vary not only histologically, but in the blood-changes to which they lead :—(1) spleno-medullary, and (2) lymphatic leukæmia. The cause is unknown. It affects men much oftener than women, and is most common during middle life.

SPLENO-MEDULLARY LEUKÆMIA.—In this variety the marrow, spleen, and liver are chiefly affected. The marrow-fat is replaced by enormous overgrowth of leucoblastic tissue, consisting mainly of myelocytes, and polymorpho-nuclear cells. Nucleated red cells are also plentiful. The

spleen is greatly enlarged, and of firm consistence. Its normal structure is lost, and closely resembles that of the marrow just described. In long-standing cases, there is marked sclerosis from increased formation of fibrous tissue. The lymph-glands are but slightly affected as a rule. As the disease progresses, there is a tendency to leucocytic accumulations in the capillaries, especially in those of the liver, causing considerable enlargement of the organ, and in the lungs.

Symptoms.—The onset is insidious. In most cases swelling, associated with local discomfort in the left-hypochondrium due to the large spleen, is the first complaint; but some begin with progressive loss of strength, or troublesome diarrhœa. However it begins, a slowly advancing anæmia takes place sooner or later, associated with weakness and emaciation, incapacity for exertion, shortness of breath, and digestive derangement. If the blood be now examined, a large increase of leucocytes will be found, amounting on an average to 400,000 per cmm., but often greater. The excess involves all the normal varieties of leucocytes, with the addition of a large number of myelocytes. The relative percentage proportions are 30–50 myelocytes, 30–60 polymorphonuclears, 5–30 lymphocytes (chiefly large mononuclear). The red cells are also reduced in number; many nucleated forms are present, and the colour-index is low.

Progressive loss of weight almost always takes place. More or less fever is also very generally observed—not persistently, but lasting for a few weeks at a time. Hæmorrhage is apt to occur, especially from the nose; but it is rarely a prominent feature. Owing to the great destruction of white blood-cells, the urine contains a large amount of uric acid, probably larger than in any other disease. Albumen is often present, with or without casts. Partly from anæmia, and partly from leucocytic accumulations in the pulmonary

capillaries, dyspnœa may be distressing. In the later stages œdema and serous effusions are not uncommon. The average duration is from one to three years, but prolonged remissions are of frequent occurrence. Death ultimately occurs from asthenia, or some complication.

LYMPHATIC LEUKÆMIA.—In this variety the morbid process begins and is most marked in the lymph-glands; but the fact should be again emphasised that the whole hæmopoietic system tends to become involved by overgrowth of lymphoid tissue. The course is for the most part shorter than in the spleno-medullary variety, and more than half the total cases are acute and rapid. The age-incidence is also younger, especially in the acuter forms. Glandular enlargement is the most striking feature, and no lymph-glands are exempt. They are usually hard, rarely tender, freely movable, and never form the large masses such as are found in Hodgkin's disease. Some enlargement of the spleen is almost always found; but, as a rule, it is moderate in degree.

The blood is pale. The red cells are progressively decreased, mainly owing to the large replacement of erythroblastic by leucoblastic tissue in the marrow; and the colour-index is low. Nucleated red cells are few or absent. The excess of white cells, though great, is less than in the spleno-medullary type—about 200,000 per cmm. as an average. The increase is made up almost entirely of lymphocytes which form from 80–95 per cent. of the whole number. Their average size is smaller in acute than in chronic cases.

Acute cases usually begin with rapidly increasing weakness, and fever; but in chronic cases, nothing may be noted for a long time beyond the glandular enlargements. When established, the symptoms are much the same as those of the spleno-medullary type, but appear earlier for the most part. Hæmorrhages, however, are considerably more common,

especially under the skin (petechiæ), and from the gums. Small nodules of lymphoid tissue in the skin are not uncommon. The affection is always fatal. Acute cases almost always die under two months, but chronic cases may live for four or five years. Older patients as a rule last longer than younger.

The only medicinal agent of any value in leukæmia is arsenic; but recently some encouraging results have been obtained in certain chronic cases by periodical exposure of the enlarged spleen to X-rays.

HODGKIN'S DISEASE.

This disease is characterised by progressive and painless hyperplasia of the lymphatic glands and of adenoid tissue in various parts of the body, associated with anæmia, but without any excess of leucocytes. The spleen is moderately enlarged in a majority of cases from lymphoid growths within it. It is most common in males. The enlargement usually begins in the glands of the neck, and progressively extends to the lymph-glands elsewhere, both superficial and deep. The rate of progress is slow, and irregular, often remaining stationary for a long time. The glands are painless, fairly soft, and freely movable under the skin to begin with; but as enlargement proceeds, and large tumour-like masses are formed by close aggregation, the glands become harder, and much less movable. But they do not fuse together, nor burst the boundaries of their capsules, nor caseate or suppurate (except as the result of some secondary infection). The liver may be enlarged—and more rarely the kidney—from adenoid growths. The number of red cells in the blood is largely reduced, and exhibit a low colour-index. As a rule the white cells are unaltered.

The leading general symptoms are a slowly progressive anæmia, fever, emaciation, and weakness, merging ultimately into a profound asthenia. The fever varies in type. Sometimes it is moderate and irregular; sometimes high and of intermittent type; or it may alternate with periods of apyrexia. The progress of a case is apt to be variously modified by symptoms due to pressure of the glandular enlargements on adjacent parts. Thus œdema of the limbs from pressure on returning veins; pain from pressure on nerves; pain, dyspnœa, and cough from intrathoracic pressure, and so on. The termination is always fatal, and survival beyond two, or at most three, years is uncommon. Death occurs from exhaustion or some complication.

Examination of the blood will easily distinguish Hodgkin's disease from lymphatic leukæmia. The only other conditions likely to give rise to confusion, are multiple glandular enlargements from tuberculosis, and lympho-sarcoma. In tuberculous cases, when the glands become painful, tender, adherent to the skin, and threaten suppuration; and when there are other evidences of tuberculosis, distinction may be easy; but cases of great difficulty are met with, in which we have to rely mainly on tuberculin-reaction tests, and microscopic investigation of an extirpated gland. In lympho-sarcoma, growth is more rapid, the glandular masses are larger, and the growth tends to invade the adjacent structures. In the early stages of any case, caution should be exercised before committal to a definite diagnosis.

With regard to *treatment*, arsenic is again the most promising remedy, but at best it is only palliative. Iron, quinine, and cod-liver oil are useful tonics. Finally, considerable improvement has been reported in certain cases by employment of X-rays.

SECTION V.

DISEASES OF DUCTLESS GLANDS.

DISEASES OF THE SPLEEN.

The spleen normally lies in the left hypochondrium in the hollow of the diaphragm, and is partly overlapped by lung, and partly in direct contact with the thoracic wall. It extends in the axillary line from the seventh rib above to the eleventh or twelfth rib below; and its anterior margin coincides roughly with the line of the ninth rib (fore part). It cannot be reached by palpation, and is only recognisable by percussion; but the dull area varies, according to the amount of distension of the stomach and the bowels, and therefore slight degrees of splenic enlargement cannot be recognised with precision. The enlarged organ extends downwards and inwards towards the pelvis and umbilicus, and may form a tumour of immense size, with great distension of the abdomen. When once it has extended beyond the lower costal border it can be reached by palpation. We find that it follows the respiratory motion of the diaphragm, that it is movable, and that it shifts its position according to the posture of the body. Its anterior edge is well marked and presents one or more characteristic notches.

The expression "diseases of the spleen" can only be employed in a qualified sense, for, so far as our knowledge goes, it would appear that all morbid conditions of the spleen are associated with or secondary to some wider

disease process, of which the splenic trouble is only a partial manifestation. Again, of these morbid conditions, few can be recognised clinically apart from enlargement of the organ; and therefore it will be convenient to group them under this head.

ENLARGEMENTS OF THE SPLEEN may be acute or chronic. Acute enlargement is a common accompaniment of many specific infections, such as typhoid, malaria, and septicæmia. It also occurs as the result of active congestion or inflammation from any cause, or infarction. It may be associated with pain or discomfort in the left side, but more often there are no subjective symptoms.

Chronic enlargement may result from mechanical venous congestion, or lardaceous degeneration, or new growth, or it may occur as a sequel of acute enlargement. It also occurs in connection with certain primary forms of anæmia, rickets, inherited syphilis, and other infantile disorders of too obscure a nature to admit of being definitely labelled. There is one form of chronic splenic enlargement associated with anæmia, which demands a few words of special reference, under the name of splenic anæmia.

Splenic anæmia is an apparently primary affection, without discoverable cause, characterised by simple hyperplasia and fibrosis of the spleen, anæmia of a secondary type, and attended in its later stages with fibrosis of the liver (*Banti's disease*). It is essentially a chronic condition, which Osler divides into three stages:—(a) simple enlargement, with little or no anæmia; (b) secondary anæmia, with a tendency to pigmentation of the skin, and hæmatemesis; and (c) a final stage characterised by jaundice and ascites. The diagnosis turns so largely on its mode of evolution, that in the early stage it is very difficult, and great care is needed before arriving at a definite conclusion. When the diagnosis is established, it would appear that removal of the spleen affords the only chance

of escaping a fatal issue ; and Osler reports three successful results in five cases of his own.

MOVABLE SPLEEN.

Occasionally the spleen drags upon its attachments, and becomes freely movable, or it may wander from its normal position. It is generally, but not necessarily, associated with enlargement of the organ. Neither is it necessarily associated with displacement of other abdominal viscera. It is more common in women, and appears to be due to relaxation of its ligaments, the tendency to which in some cases is hereditary. The patient may be quite unaware of the condition, or may complain of local dragging sensations which are relieved by the recumbent posture. In marked cases there is a possible danger of the spleen twisting on its ligaments, and so becoming strangulated. This is a serious condition, and demands prompt surgical intervention. Sometimes again the organ may become fixed by adhesions in an abnormal position, and give rise to difficulty in diagnosis. No treatment is required in slight cases, beyond wearing an abdominal belt ; but in bad cases, fixation by surgical means becomes necessary.

DISEASE OF THE SUPRA-RENAL GLANDS.

The supra-renal bodies, or adrenals, are highly vascular ductless glands, closely connected with the sympathetic nervous system, both in their origin and nerve-supply. They yield an internal secretion (adrenalin) which has, among other possible effects, the striking property of raising the blood-pressure by increase of cardiac contractility and arterial constriction. Adrenalin is produced exclusively in the medullary portion of the glands, by cells of a special type, known as *chromaffin-cells*, yielding an

intense brown colour on staining with chromic salts. It is said that chromaffin-cells are also present in the sympathetic ganglion and elsewhere, but the adrenals are undoubtedly their principal seat. It is certain that the glands in question have important functions, closely connected with their internal secretion, which are liable to disturbance in many ways by infective conditions, inflammation, or new growths affecting them; but the present state of our knowledge does not admit of differentiation beyond what is known as Addison's disease now about to be described.

ADDISON'S DISEASE.

Addison's disease is a general affection which is mainly characterised by general debility and bronzing of the skin. It is due to progressive inadequacy and ultimate arrest of adrenal function, which depends in a large majority of cases upon tuberculous disease of the supra-renal glands. Degenerative changes in the sympathetic nerves and ganglia of the upper abdomen are found in a certain proportion of cases. The affection is relatively rare, but occurs most often in males between thirty and forty years of age.

The *symptoms* may be ranged in three main groups—general and progressive debility, pigmentation of the skin, and gastro-intestinal disturbance.

Gradually increasing weakness is usually the earliest indication of trouble, shown by unusual fatigue after physical or mental exertion. This is often associated with general apathy, inertness, and lack of interest in affairs. Though he may not look ill, the patient complains of feeling always tired; and slight overstrain of any kind is sometimes sufficient to cause quite alarming faintness and prostration. The general lack of tone is also betrayed by the low-pressure frequent pulse, clammy sweats, with palpitation and dyspnoea on the least exertion. Sleepless-

ness and headache are of common occurrence. Meanwhile pigmentation of the skin develops, and may, in some cases, be the first thing to attract attention. It varies in colour from a pale brown to a dark bronze. In typical cases it is diffuse, but better marked on parts exposed to pressure, and on normally pigmented parts such as the areolæ of the nipples, axillæ, and pudenda. Similar pigmentation is often found on the lips, and buccal mucous membrane. Anæmia is not a marked feature, and no characteristic changes in the blood are met with. In many cases (but not in all) there are indications of gastro-intestinal disturbance, such as pain, loss of appetite, spontaneous nausea and vomiting, and irregular action of the bowels.

Cases vary considerably in the relative prominence of different groups of symptoms, and also in rapidity of development. Remissions not infrequently occur, but are only of temporary duration, the progress being invariably towards a fatal termination. Death ultimately occurs from asthenia or syncope, and is sometimes preceded by grave nervous disturbance.

Before arriving at a diagnosis, care should be taken to exclude brown pigmentation of the skin such as may be met with in elderly persons of dirty habits, especially when associated with pediculosis; in pregnant women; in chronic malaria; in chronic tuberculous or malignant disease of the peritoneum; in some forms of chronic jaundice; and in those who have been submitted to a long course of arsenic.

TREATMENT.—In a certain number of cases, the internal administration of supra-renal extract, in tabloids containing gr. j. thrice daily, may be of service, but more often it fails. Every endeavour should be made to keep up the strength by tonics, cod-liver oil, wine, good food, warmth, and fresh air.

DISEASES OF THE THYROID GLAND.

The thyroid gland consists of follicles enclosed in a fibrous capsule, and lined by cubical or columnar cells, yielding a colloid material, the most characteristic constituent of which is an organic compound called "iodothyryn," containing a considerable but variable quantity of iodine. Much obscurity still prevails as to the behaviour and functions of the thyroid; but it is clearly essential to life, since its complete removal is followed by death; whereas, if a small portion be left, the consequences of removal may be escaped. It is also proved that its influence is exerted by an internal secretion, since the effects of removal or arrest of function by disease can be averted by supplying an extract of the gland, artificially prepared.

With diminished thyroid activity, growth is lessened, the skin and hair show impaired growth, the nervous system degenerates, and vital processes generally are depressed. On the other hand, when thyroid activity is exaggerated, there is increased metabolism, and consequent loss of weight, cardiac excitement, nervous irritability, and sleeplessness.

The thyroid is liable to secondary disturbance in the course of many acute infectious diseases. In girls, also, simple temporary enlargement of the gland often occurs in connection with menstrual excitement and certain hysterical conditions. But, apart from these relatively unimportant affections, we meet with cases of a primary nature, which will now be described.

SIMPLE GOITRE.

Here we have to deal with a parenchymatous hypertrophy of the gland, which is apt to undergo cystic transformation.

It is practically confined to women, and occurs either in a sporadic or epidemic form. For reasons as yet unknown, it is especially prevalent in the hilly parts of Derbyshire, in the Swiss Alps, and in other mountainous regions. It is attended with no constitutional disturbance, and risk only arises from compression of the trachea, or pressure upon veins. It is therefore chiefly of surgical interest.

EXOPHTHALMIC GOITRE.

Exophthalmic goitre, otherwise known as Graves' or Basedow's disease, is characterised in typical cases by thyroid enlargement, prominence of the eyeballs (exophthalmos), and cardio-vascular excitement. It is far more common in women than in men, especially in those of a nervous temperament. It appears sometimes to be induced by nervous shock, fright, or violent emotion, or nervous overstrain. Its pathology still leaves many points to be cleared up, but there is a strong presumption that it represents thyroid secretory activity in excess; but whether this is brought about by excessive secretion, modification of its normal composition, or readier access to the general circulation is not definitely ascertained.

The *symptoms* may come on suddenly; but, more often, the onset is quite gradual. The first complaint is usually of palpitation, with undue frequency of the pulse, which is much exaggerated by excitement of any kind. The area of cardiac pulsation is increased, and functional cardiac murmurs are of frequent occurrence. The superficial arteries throb visibly, capillary pulsation is also readily seen, and widely distributed erythematous flushing is often present.

After a while *prominence of the eyeballs* is noticed, and this gradually increases until at last the eyelids are no longer competent to cover the eyes completely, and con-

sequently the eyes are apt to become inflamed. In certain cases, on turning the eyes downward, the upper eyelid lags (von Græfe). *Enlargement of the thyroid* may be uniform and general, or only affect one lobe; and generally develops quite slowly. The gland is soft, elastic, and pulsatile, and a systolic thrill may be felt, or a murmur heard over it. There is always evidence of more or less nervous disorder, usually in the form of irritability, excitability, and general restlessness. Sometimes we meet with giddiness, complaints of tingling or throbbing, but headache is not a common symptom. Fine *tremor* is very generally observed. Anæmia, emaciation, irregularities of temperature, and gastro-intestinal derangement are common accompaniments; and paroxysmal dyspnœa, generalised or patchy pigmentation of the skin, œdema, and polyuria with slight albuminuria, may also be observed. Death is a rare termination, yet complete recovery is exceptional; and though acute cases have been recorded, the usual course is excessively chronic.

In the *treatment* of this affection, rest and freedom from excitement are essential. The diet should be plain but nourishing, and alcohol, tea, and coffee should be forbidden. The medicinal treatment is very unsatisfactory and uncertain. The best results are perhaps obtained by a course of arsenic and strophanthus or convallaria, in conjunction with ice applications to the neck. Bromides are useful with marked nervousness and tremors. There is much evidence in favour of galvanism of the neck. Only weak currents should be used. The positive pole should be fixed to the nape of the neck, while stroking movements are made along the front border of the sterno-mastoid with the negative pole. Prolonged change of surroundings, in a clear bracing climate, is sometimes of the greatest benefit. In severe cases, the question of removal of part of the gland by operation is well worth consideration.

MYXEDEMA.

This disease is due to loss of thyroid function, and is characterised by subcutaneous swelling and mental failure, and depends upon atrophy of the thyroid gland. It is most common in married women from 35 to 50 years of age. The dependence of the disease upon thyroid inadequacy is conclusively proved by its artificial production in animals after extirpation of the thyroid gland, and by the specific results of treatment by thyroid extract. The gland is generally small, atrophied, and fibrous, but occasionally destruction of the proper tissue of the gland is associated with enlargement. The subcutaneous swelling is due to the formation of tissue resembling granulation tissue, infiltrated with gelatinous material, formerly believed to be mucin, but with doubtful accuracy. Degenerative changes have been found in the central nervous system, and arterio-sclerosis is not uncommon.

SYMPTOMS.—The onset is insidious, but when the disease is fully developed the condition is characteristic. There is a general puffiness of the body, which consequently increases in weight. The skin is dry and infiltrated, but does not pit on pressure. The face is rounded, and the eyelids, nose, and lips are thickened. Similar swelling affects the tongue and soft palate. The scalp becomes scurfy, the hair falls out, the nails are brittle, and the teeth may become loose. Bodily movements are languid, and speech is slow and blurred. Memory is feeble, the mind is dull, and the temper is irritable. The patient is pale, and has all the other symptoms of anæmia, the temperature is subnormal, and the pulse is weak. The appetite is bad, and the bowels are costive. The urine is abundant, of low specific gravity, with a marked diminution of urea.

The course is very chronic, often extending over many years. A cure may be effected and sustained by treatment,

but relapse occurs when the latter is suspended. If left alone, the patient ultimately sinks from exhaustion, or intercurrent disease.

TREATMENT.—The present treatment of myxœdema forms a new departure in therapeutics. It consists of the introduction into the system of an active principle derived from the thyroid gland. This may be done, either by the subcutaneous injection of a liquid extract, in doses of $\mathfrak{m}\text{x.}$ to $\mathfrak{m}\text{xx.}$, once or twice a week; or, what is far better, by the internal administration of gr. ij. to v. of the dried extract daily. The latter is obtainable in the convenient form of tablets which keep well, and are always ready to hand.

CRETINISM.

This term is applied to myxœdema occurring sufficiently early in life to interfere with normal development. In its most typical form it is due to congenital absence of the thyroid gland; but milder forms are occasionally observed where the gland has atrophied, as a sequel of some acute specific fever contracted in early childhood. The condition is not usually recognised until the child is over a year old, when it becomes apparent that development is not proceeding in a normal way.

When established, the appearance of the child is very characteristic. The body is short and stumpy; the fontanelles remain open; the face is broad and puffy; the nose flattened; the margins of the eyelids and nostrils are thickened; dentition is delayed; and no progress is made with walking or talking. The abdomen is large and prominent, and the limbs are podgy. In a word, physical and mental development are at a standstill; and, if not relieved by treatment, the child lapses into idiocy. Curious pads of fat may often be noted in the supra-clavicular fossæ which have considerable diagnostic importance. The principles of treatment are the same as for myxœdema in adults.

SECTION VI.

DISEASES OF THE RESPIRATORY SYSTEM.

GENERAL CONSIDERATIONS.

STRUCTURAL CONDITIONS.—The lungs consist essentially of a mass of sacculated chambers (infundibula and air-cells) with thin, elastic, and highly vascular walls, probably representing a respiratory surface of over 200 sq. yds. By a series of branching tubes (trachea and bronchi) the air-cells are brought into direct communication with the outer air through the mouth and nose. The infundibula connected with each terminal bronchus (bronchiole) form clusters or lobules, which are bound together by connective tissue. The bronchi contain cartilaginous plates, muscular fibres, and mucous glands; and are lined by columnar ciliated epithelium for the greater part of their course. The terminal bronchioles, however, have no cartilage or mucous glands, and are lined by flattened non-ciliated cells. The bronchi are supplied by the bronchial artery, the blood of which is returned by the bronchial veins, and thus belongs to the systemic circulation; but the capillaries of the terminal bronchioles mingle with those of the air-cells, and discharge into the pulmonary vein. The pulmonary circulation proper is supplied by the pulmonary artery, which accompanies and branches with the bronchi, breaks up into capillaries in the walls of the air-cells, and returns by the pulmonary veins which take a separate course in the interlobular connective tissue.

The nerve-supply of the lung is derived from the anterior and posterior pulmonary plexuses formed by fibres from the vagus and sympathetic. The lymphatics begin in the interlobular connective tissue and bronchial mucous membrane, and drain into larger vessels leading to glands in the mediastinum. Those on the surface of the lung communicate with those of the visceral pleura, a fact which has an important bearing on the transfer of infection between the lungs and pleura.

MECHANISM OF BREATHING.—Under normal conditions, the lungs are held in a state of permanent moderate inflation; and, being elastic, exert a negative pressure upon the chest-wall, amounting to about 5 mm. Hg. at the end of expiration, and 7 mm. at the end of inspiration. Accordingly, when air or fluid enters the pleural cavity, the negative pressure is reduced or abolished, and both the chest-wall and lung recoil, provided they retain their normal elasticity, and there is nothing to restrain their freedom of action. This fact not only explains various physical signs of pleural and pulmonary disease, but it also throws light upon the mechanism of breathing. While inspiration essentially depends upon enlargement of the chest-cavity by contraction of the diaphragm, and various muscles attached to the ribs, it is clear that in ordinary quiet inspiration, the requisite effort will be considerably reduced by the elastic recoil of the ribs. Ordinary expiration is an entirely passive event, due to elastic recoil of the lungs on cessation of inspiratory muscular contraction; but forced expiration involves effort, chiefly on the part of the abdominal muscles in forcing the diaphragm upwards. The respiratory muscles are governed by a nervous centre in the medulla, which is automatic in its rhythmical action, but largely influenced by the character of the blood supplied to it, and by various peripheral impressions conveyed to it from elsewhere.

PHYSICAL SIGNS.—The art of physical examination of the chest can only be taught at the bed-side, and learnt by personal practice and experience; but a few remarks on the subject may not be out of place here.

The methods commonly employed for this purpose are inspection, palpation, percussion, and auscultation. By *inspection* we note the size and development of the chest, its shape and movements, together with the presence or absence of pulsation, tumours, large veins, etc. *Palpation* is chiefly useful for the confirmation of the results of inspection, and for estimating the vibration of the chest-wall on phonation (vocal fremitus). The value of *percussion* rests upon the fact that by this means we can not only indicate the limits of air-containing organs or tissue, but roughly measure their relative air-content, by comparison with standards established by experience. By *auscultation* the intensity, rhythm, and quality of the breath sounds are observed, the vocal vibration of the chest-wall is estimated (vocal resonance), and the presence or absence of adventitious sounds is noted.

In order to make the most of this source of information it is desirable always to proceed according to a regular definite system, taking each method of examination in order, and making observations under each in turn. If this plan were uniformly adopted, mistakes of omission would be much less frequent than they are. It seems also desirable to point out—though it savours of a truism—that only conclusions of a physical nature can be drawn from a physical observation. A physical observation is one thing, its clinical interpretation is quite another; and the beginner will be well advised in clearly separating the two things in his mind. For instance, supposing a friction-sound is heard in some part of the chest, all that it implies is the physical fact of movement upon each other of rough surfaces in contact. Its reference to pleural inflammation

is quite another line of argument, resting upon quite different data.

Careful analysis of the physical conditions underlying individual signs will add greatly to the interest and helpfulness of physical examination. Thus, in the instance just given, we see that the three elements of movement, roughness, and contact are essential. If either is absent, no friction-sound will be heard. It follows also that (other things being equal) it is likely to be first and best heard where the movement of the chest-wall on the lung is greatest—namely, at the ends of the lower true ribs; that it will not occur till the pleura becomes rough from exudation; and that it will cease as soon as the surfaces are separated by effusion.

It may be worth while to give yet another example. In regard to quality, all breath-sounds fall into one of two main groups—vesicular or bronchial. Both types alike present many sub-varieties, but the root-difference between them is as follows. By common consent, vesicular breathing, in any of its varieties, consists of a fundamental glottic sound, modified by the to-and-fro passage of air in the air-sacs. The cause of this does not for the moment concern us. It suffices to say that the necessary and essential points are that not only should air-cells be present, but that air should be passing to and fro in them. In bronchial breathing this vesicular modification is lacking, either because in that part of the lung the air-cells have been destroyed—*e.g.* by fibroid change; or that they are filled with exudation, as in acute pneumonia; or that they are collapsed and uninflatable; while in each case the bronchi of the part remain patent. The direct inference thus to be drawn from bronchial breathing is that, though air reaches the part, either that the air-cells are absent, or that for some reason air cannot enter them.

Dyspnœa.—This term is applied both to rapid and labo-

rious breathing; and in either case we speak of the dyspnœa as objective. In certain cases, however, notably of hysteria, a sense of air-hunger may be felt without any outward disturbance of breathing, and this is distinguished as subjective dyspnœa. All cases of marked objective dyspnœa are accompanied with subjective dyspnœa; but in slighter cases, as the result of an equilibrium which gradually becomes established between the supply of oxygen and elimination of carbon dioxide, the former may occur without the latter. The depth of respiration is generally (but not always) in inverse proportion to its frequency. As a rule, dyspnœa is the proximate result of an abnormal condition of the blood which is supplied to the respiratory nervous centre in the medulla. Thus, dyspnœa occurs (1) when the blood is over-heated, as in fevers; (2) when it contains unusual ingredients, as in some cases of uræmia; (3) when it contains an excess of carbon dioxide or is deficient in oxygen, as a result of anæmia, disease of the lungs or heart, obstruction of the air-passages, mechanical interference with the movement of the chest-walls or diaphragm, paresis or spasm of the respiratory muscles. In other cases dyspnœa results from general nervous excitement, or from reflex irritation of the respiratory nervous centre due to such causes as enlarged tonsils, nasal polypus, or worms.

Sometimes the difficulty mainly attaches to inspiration as in certain forms of laryngeal paralysis; at other times to expiration, as in emphysema; but more often it affects both inspiration and expiration. When dyspnœa is so severe that breathing is only possible in a more or less upright posture, it is termed *orthopnœa*. When the difficulty in breathing comes on in more or less sudden attacks of temporary duration, it is said to be *paroxysmal*. In all cases of partial obstruction or constriction of the laryngeal passage, the breath-sounds have a peculiar harsh wheezing

quality, known as *stridor* or *stridulous breathing*. Another variety, known as *Cheyne-Stokes respiration*, is characterised by a series of respirations gradually increasing in depth up to a maximum, then gradually declining to a minimum, and followed by a considerable pause. It is a sign of very unfavourable import.

Cough.—Coughing consists of a long-drawn inspiration followed first by closure of the glottis, and then by one or more forcible expiratory efforts by which the glottis is forced open with noise. It is a purely reflex act, the afferent impulses of which travel upwards for the most part through fibres of the vagus nerve coming from the throat, air-passages, lungs, stomach, or ear. Irritation of any of these parts is therefore liable to be attended with cough. Cough may be simple or paroxysmal; as to quality, it may be hacking, hoarse, barking, metallic, aphonic, and so on; and it may be dry, or accompanied with secretion. When associated with irritability of the pharynx, cough is generally paroxysmal and apt to lead to vomiting.

Expectoration.—In health, the secretion of mucus in the air-passages is small in amount, and there is no need for expectoration. But when increased as the result of inflammation, or when serous exudation, blood, or pus is present in the air-passages, cough is almost always attended with more or less expectoration, except in young children. Much may be learnt from observation of the quality, consistence, character, and odour of the sputa. As a general rule, abundant sputa are of bronchial origin. The consistence varies greatly, from the watery sputa of bronchorrhœa and œdema of the lungs, to the tenacious viscid sputum of certain forms of pneumonia. As to character, the sputa may be mucoid, muco-purulent, purulent, or bloody; airless, or frothy; confluent, or in separate rounded plugs floating in a mucoid fluid (*nummulated sputa*)

which are suggestive of origin in a cavity. As to odour, they may be odourless, offensive, or gangrenous. Branched fibrinous casts of the bronchi are occasionally met with; and echinococcus cysts are sometimes discharged in the sputa. On microscopic examination, fibres of elastic tissue may be found, as the result of destruction of lung-tissue, especially in phthisis. The best way of detecting them is by boiling the sputa in caustic potash, diluting with water, and allowing to stand, when the fibres will be found in the sediment. Bacterial organisms of various kinds may also be discovered by microscopic examination after appropriate staining.

SUB-SECTION I.

DISEASES OF THE NOSE.

THE nose, besides being the organ of smell, has other important functions. It forms the normal entrance to the respiratory passages; it warms, moistens, and filters the air on its way to the lungs. It not only arrests the progress of microbic particles in transit, but its natural secretions seem to have distinct bactericidal effects; so that atrophic rhinitis, where such secretions are wanting, is marked by the offensive smell of decomposition. Moreover, the nose plays an important part in determining the quality of articulation. Among troubles of nasal origin, those which occur in connection with nasal obstruction first require notice.

Nasal obstruction may arise in several ways—by simple swelling or thickening of the mucous lining, by adenoid and other growths, by impaction of foreign bodies, and by congenital malformations. As the result of relatively smaller nasal passages, obstruction more readily occurs in

young children, and gives rise to more striking symptoms. When it is marked and persistent, the patient breathes through his mouth, which is kept slightly open. The voice is thick and stuffy, with a special difficulty in the vocalisation of nasal consonants. As the air is neither warmed nor filtered as it enters the mouth, a dry irritable condition of the throat is set up, with special liability to inflammation, which may involve the Eustachian tubes, causing deafness and other ear troubles. Such inflammation may extend to the remoter air-passages, provoking hoarseness, with a tendency to frequent "colds" or croupy attacks. Sleep may be restless and disturbed by dreams, and attended with snoring. Amongst young children, chronic nasal obstruction is a common cause of malnutrition, headaches, inaptitude for mental work, and defective chest development, and is apt to be overlooked.

NASAL CATARRH.

Acute nasal catarrh or coryza is most frequently part and parcel of a simple catarrh from chill or "*cold in the head.*" In many cases it is undoubtedly contagious, and probably depends upon some microbe. Coryza is also met with as a symptom of other diseases, or as a result of local irritation. As a primary affection the onset is characterised by chilliness followed by mild fever, general malaise, with pains in the back and limbs. Sneezing then occurs, attended with the discharge of thin clear fluid from the nostrils. The latter are obstructed, the eyes are red and weeping, the articulation of nasal sounds is difficult, and the sense of smell (and with it of flavours) is dulled or lost. An herpetic eruption on the lips is also common. The inflammation may extend to the Eustachian tube, causing impairment of hearing; to the throat, causing soreness; or to the larynx and trachea, causing huskiness and cough. In a day or two the discharge becomes muco-

purulent and more abundant, and later on the symptoms gradually subside. In frequently recurring catarrh, the possibility of a nasal or post-nasal growth should always be kept in view.

A hot bath, followed by a moderate opiate and a little spirit and hot water at bedtime (on an empty stomach), with a saline purge next morning, is the best treatment in the initial stage. When the catarrh is established, saline diaphoretics are indicated. If there be much fever, drop doses of tincture of aconite every hour are of great service, especially in children. Inhalations of steam, medicated with carbolic acid or camphor, or the employment of a spray of a two or three per cent. solution of cocaine, often give great relief. During convalescence, tonics are required.

CHRONIC NASAL CATARRH may be a sequel of previous acute attacks; and in weak states of health, catarrhal inflammation of any kind is often difficult to shake off. It is frequently associated with hypertrophy of the nasal mucous membrane, or with post-nasal adenoid growths. The latter is one of the commonest causes of chronic nasal catarrh in young children, causing them to breathe through the mouth, and to snore in their sleep; and giving rise to stuffy articulation, mental dullness, and more or less deafness.

There is another form of chronic nasal catarrh, characterised by atrophy of the nasal mucous membrane, in which the discharge is quite scanty, and dries on the inner surface of the nostril, forming flakes, which decompose and emit a disgusting odour (*ozæna*). The patient fortunately loses his sense of smell. *Ozæna* also occurs in connection with ulceration of the nostril arising from syphilis, caries, or impacted foreign bodies.

As to *treatment*, unless the cavities are extremely irritable, washes or injections are demanded. Dilute tepid

solution of bicarbonate of soda and borax (about gr. v. of each to the ounce) is of most service. If special detergent effects are required, the addition of a little carbolic acid is desirable. Sprays may be substituted for injections if the latter are not tolerated, but they are less effective.

Constitutional treatment of a tonic kind is very necessary, with residence in a warm and dry climate if possible. The possibility of nasal or post-nasal growths should not be overlooked.

HAY FEVER.—Hay fever is a special form of coryza, which, in those predisposed to it, tends to recur every year—generally in the early summer. It is frequently associated with bronchitis and asthmatic attacks. The affection is more common in the young than the old, in women than in men, and as a rule is associated with the neurotic temperament. It is caused by the inhalation by the nose of the pollen of grasses and certain other plants, which is said to yield a toxin of an extremely irritating nature.

One of the best prophylactics is residence in a dry, bracing climate, for everything which tends to invigorate the system reduces the liability to attacks. The local measures recommended for acute nasal catarrh are very useful; but for severe cases, electro-cauterisation of altered mucous membrane may be beneficial.

SUB-SECTION II.

DISEASES OF THE LARYNX.

ORGANIC diseases of the larynx include acute and chronic inflammation (with the conditions to which inflammation gives rise, such as ulceration, cedema, and perichondritis), and new growths.

Certain local phenomena are more or less common to all laryngeal affections. (1) *Alterations in the voice*. True vocal sounds originate in the larynx, and are due to concurrence of an expiratory effort with approximation, tension, and vibration of the vocal cords. From feebleness of the respiratory act, the voice becomes faint and weak; and from uncertainty in its production, it becomes tremulous. Should the approximation, tension, or vibration of the cords be prevented by paralysis, inflammation, ulceration, or new growths, hoarseness (*dysphonia*), or actual loss of voice (*aphonia*) occurs. Temporary variations in the vocal *pitch* also result from alteration in the tension of the cords; and therefore relaxation from any cause deepens its tone. The *articulate quality* of the voice is mainly related to the physical condition of the throat, nose, and mouth, and thus is modified by enlarged tonsils, paralysis of the soft palate, nasal obstruction, and interference with the movements of the tongue. (2) *Dyspnœa*. This is noisy, hissing, or stridulous, in proportion to the amount of obstruction in the air-passages by which it is caused; and is often accompanied with marked depression of the larynx during inspiration. Sudden attacks of a paroxysmal nature are frequently met with, as a result of spasmodic closure of the glottis. Inspiration and expiration are equally affected when the obstruction is material and persistent; but in certain paralytic and other conditions, either inspiration or expiration may be alone affected. (3) *Cough* due to laryngeal irritation is frequent, paroxysmal, and of a harsh, hoarse, or barking quality. (4) The *expectoration* in recent inflammation is generally scanty and viscid; but in chronic laryngeal affections it is more abundant, muco-purulent, or it may be streaked with blood. (5) *Dysphagia*, or difficulty in swallowing, is not a common symptom of ordinary laryngitis; but is apt to occur when the epiglottis is the seat of disease, especially in connection with ulceration.

(6) *Pain* is frequently present, and varies from mere uneasiness and a sensation of tickling about the throat, to burning irritation; and it is sometimes accompanied with external tenderness on pressure of the adjacent parts.

ACUTE CATARRHAL LARYNGITIS.

Among the most frequent causes of acute catarrhal laryngitis are (*a*) catching cold, (*b*) the introduction into the larynx of irritating vapours, acid liquids, or water, (*c*) over-straining the voice, (*d*) morbid growths, and (*e*) ulceration. It also occurs as a frequent symptom of many acute diseases, such as scarlet fever, measles, typhoid, and small-pox. Previous attacks, enlarged tonsils, post-nasal growths, and sedentary indoor occupations may be regarded as predisposing causes. It is more common and more serious in children than adults.

SYMPTOMS.—As a rule (but not invariably) the disorder begins with the signs of an ordinary cold. There is more or less febrile disturbance accompanied with hoarseness of the voice and cough, sometimes increasing to aphonia. Burning or tickling sensations in the larynx may be complained of, and the throat may be sore. The character of the expectoration—if there be any—varies greatly. With the laryngoscope, the mucous membrane is seen to be red and turgid. In children, from the small size of the laryngeal passage and the greater laxity of the mucous membrane, there is relatively more swelling, and consequently more interference with breathing, than in adults. Children are also especially prone to acute paroxysms of dyspnœa and stridor from spasmodic closure of the glottis. Spasmodic laryngitis has considerable resemblance to membranous croup, for which it is liable to be mistaken. The attack comes on suddenly at night with stridor and alarming inspiratory dyspnœa, but it is of short duration as a rule. On cessation of the spasm the child usually falls

asleep, and is much better during the following day. A croupy, clanging cough and some catarrhal symptoms persist for a few days, with more or less spasm by night, but with diminished severity, until recovery takes place. Enlarged tonsils and post-nasal adenoids specially predispose to spasmodic laryngitis; and, after one attack, recurrence may result from any slight cold. It is rarely fatal.

Acute œdema of the larynx is also an occasional and dangerous complication of catarrhal laryngitis; but it happens most often in connection with scarlet fever, erysipelas, diffuse inflammation of the neck, and the introduction of scalding water into the larynx. The œdema mainly involves the loose tissue of the ary-epiglottic folds, which may become so swollen as entirely to block up the entrance into the larynx. Under these circumstances, severe dyspnœa is inevitable, and, if not relieved, death from asphyxia soon follows. Thus although acute catarrhal laryngitis usually ends favourably, yet dangerous complications may occur which demand prompt attention. Sometimes it passes into the chronic form.

ACUTE MEMBRANOUS LARYNGITIS—CROUP.

Croup is a peculiarly fatal disease of childhood, which is characterised by membranous laryngitis. Most cases are undoubtedly of a diphtheritic nature, but this is not true of all. The only means of distinguishing those cases which are diphtheritic from those which are not, is by concurrent exudation on visible parts of the throat, or by discovery of diphtheria bacilli on examination of the secretions of the throat.

It is essentially a disease of childhood, generally occurring between the ages of two and seven years, and in boys more often than in girls.

MORBID ANATOMY.—The affected mucous membrane is

at first red and swollen. The epithelium is shed, and a layer of coagulable lymph is then thrown out on the surface, forming a false membrane. The latter varies in thickness from a mere film, to two or three lines or more, and is more or less adherent. Gradually it becomes loosened, and in favourable cases may be partly expelled by coughing, and the parts may gradually return to their normal condition. The inflammation and false membrane may extend for a variable distance into the pharynx above and the trachea below, while beyond this point it gradually merges into inflammation of a catarrhal type.

SYMPTOMS.—Signs of an ordinary catarrh, slight fever, and sore throat usually precede the laryngeal symptoms for a short time, but sometimes the onset is more abrupt. The first indication of laryngeal mischief is huskiness of the voice, which becomes *progressively* more marked till it is reduced to a whisper. Cough quickly follows, at first hoarse, clanging, and paroxysmal; but as the disease progresses, the paroxysmal character is lost and it becomes almost or quite toneless. Meanwhile, the breathing becomes frequent, difficult, and stridulous. Inspiration is first affected, but afterwards expiration becomes equally difficult and laborious. The *alæ nasi* are in active motion, the epigastrium and lower part of the chest sink in with each inspiration, the face and general body-surface become dusky and livid; and the child tosses about, clutching at his throat as if to remove some obstacle there situated. Suffocative attacks—sometimes fatal—are apt to occur from time to time, especially at night, as the result of accumulated secretion or spasm of the glottis. Viscid mucus with fragments of false membrane may be expelled by coughing, which gives temporary relief. The temperature is raised—sometimes considerably; the strength is gradually diminished; the skin is dry and hot at first, but as prostration supervenes it becomes cold and clammy; the pulse is

frequent, and ultimately small and feeble; the glands at the angle of the jaw may be somewhat swollen and tender; and the urine usually contains albumen.

The disease progresses in this fashion for some four or five days, when in favourable cases—possibly after the expulsion of false membrane—the cough becomes looser and louder, the breathing easier, and recovery sets in slowly. In fatal cases, which are less common under modern methods of treatment than formerly, the symptoms become steadily worse, until death occurs from profound asthenia or asphyxia. Death is most frequent on the fourth day.

By means of auscultation, information may be gained as to the amount of air entering the lungs. The *breath-sounds are weak in proportion to the obstruction*, but they are often masked by the laryngeal sounds. Sonorous and mucous râles are usually present, indicating bronchitis. Broncho-pneumonia and pulmonary collapse are also common complications.

TREATMENT.—All cases of acute laryngeal inflammation require rest in bed, warm clothing, a temperature of 70° F. and *air moistened by steam*.

In *adults*, inhalations of steam, to which tr. benzoin. co. 3 j., or succus conii ℥ ij. with sodæ carb. gr. xx., may be added, are very soothing. Hot sponges, or linseed and mustard poultices applied externally to the throat, may often be used with advantage. The skin should be excited to action by warm water or vapour baths, warm drinks, and diaphoretic salines; and the bowels should be well opened. At the outset, a saline such as liq. ammoniæ acetatis, in combination with tinct. camph. co. is useful to relieve the cough; and for troublesome irritability of the larynx, inhalation of the fumes of ammonium chloride, or a cocaine spray may be tried. All smoking of tobacco should be forbidden.

In acute œdema of the larynx hot sponges should be

applied to the front of the neck, and ice should be sucked. In urgent cases, after swabbing with a 5 per cent. solution of cocaine, the swollen part should be scarified. In some cases, tracheotomy may be necessary.

In acute spasmodic laryngitis of *children*, great relief may be given during the attack by the application of a succession of hot sponges to the throat, and by the administration of ʒj. doses of ipecacuanha wine, or gr. $\frac{1}{4}$ – $\frac{1}{2}$ of sulphate of copper, every quarter of an hour, till vomiting occurs. The room should be kept warm but well ventilated, with a steam-tent over the child's cot. Tracheotomy is seldom required. Between the attacks, the child may be given some simple saline with a little ipecacuanha wine and paregoric; and turpentine liniment may be rubbed into the chest with advantage. The diet should be light and nutritious. During convalescence, a general tonic regime with change of air is recommended; and steps should be taken for the removal of enlarged tonsils or adenoids, if present.

In *all* cases of membranous laryngitis, whether proved to be diphtheritic or not, subcutaneous injections of diphtheria antitoxin should be employed, as already described in the section on diphtheria. From the extreme tendency to prostration, a good supply of nourishing food is demanded, especially during the early stages, and stimulants are often required from the outset. For the same reason lowering treatment of any kind must be avoided. Emetics such as gr. $\frac{1}{4}$ – $\frac{1}{2}$ of sulphate of copper at short intervals may be resorted to with advantage when the glottis is blocked by secretion and exudation. A spray of hydrarg. biniodide solution (1 in 1000) or carbolic acid solution (1 in 80) may be employed for the throat with great advantage. Lastly, tracheotomy must not be deferred—as too often happens—until the patient is practically moribund, but should be promptly performed

on the failure of the preceding measures to relieve the dyspnœa.

CHRONIC LARYNGITIS.

Chronic laryngitis may occur as a sequel of the acute form; or it may depend upon over-straining of the voice, the inhalation of irritating matter, new growths, syphilis, tubercle, chronic alcoholism, or the abuse of tobacco.

Whatever be the cause, the *symptoms* are those common to laryngeal mischief of all kinds, as already described. They vary only according to the seat of the lesion, and the physical changes induced by it. The employment of the laryngoscope is therefore essential for a proper diagnosis.

Simple chronic catarrh causes but little discomfort in the larynx. The laryngoscopic changes are those of congestion and swelling of the mucous membrane, or vocal cords.

In "*Clergyman's sore throat*" the racemose glands of the larynx and follicles of the pharynx are enlarged and congested. The inflammation either extends from the larynx to the pharynx or *vice versa*. The symptoms are usually milder than in simple catarrh, but often very intractable.

New growths in the larynx may be either malignant or benign. Epithelioma among the former, and papilloma and fibroma among the latter, are by far the most common. They can only be diagnosed with certainty by means of the laryngoscope.

Syphilis.—During the secondary stage it is rare to meet with other than a catarrhal type of laryngitis; but in the tertiary stage, there may be destructive ulceration, the cicatrisation of which is very apt to be attended with serious constriction of the larynx, and more or less distortion of its parts. Tertiary ulcers are especially prone to attack the epiglottis.

Tuberculous laryngitis.—The larynx suffers in a majority

of cases of phthisis, but more often the affection is of a simple catarrhal nature. A patchy distribution of catarrhal changes is more significant of tuberculous origin than when uniformly distributed. Among the more characteristic appearances are ulceration—most frequently situated on the posterior part of the vocal cords; and pale localised swellings, which may affect the epiglottis, ary-epiglottic folds, the inter-arytenoid space, or ventricular bands. Pyriform swelling of the ary-epiglottic folds is especially characteristic. These swellings may ultimately break down and ulcerate. *Necrosis of the cartilages* more often results from laryngeal phthisis than from all other causes put together, and is generally associated with ulceration. Pus forms and collects under the perichondrium, and the cartilages necrose and may even be expectorated in fragments. The prognosis of laryngeal phthisis is most unfavourable.

GENERAL TREATMENT OF CHRONIC LARYNGITIS.—All varieties urgently require rest for the larynx, a dry air of equable temperature, warm clothing, avoidance of exposure, and removal of any source of irritation. A course of mercury and potassium iodide is indicated in the syphilitic variety. When deglutition is very painful or difficult, only liquids thickened with cornflour or arrowroot should be given, and in some cases nutrient enemata may be necessary. Further relief may be given by steam inhalation; or by the use of a cocaine spray shortly before food.

Local remedies may be employed in the form of inhalations of vapour or atomised spray, or liquids applied directly by means of a special brush. Various formulæ will be given in the Appendix. As many of these applications are attended with considerable temporary pain, a preliminary spray or application of cocaine is generally desirable. In extreme cases tracheotomy may be required;

and certain forms of new growths need removal by operation.

LARYNGISMUS STRIDULUS.

Laryngismus is a disease characterised by spasmodic approximation of the vocal cords, constricting the glottis, and hence giving rise to dyspnœa and stridor.

This affection is rarely met with except in association with rickets. The attack is of reflex origin, generally in connection with gastro-intestinal irritation, dentition, or nervous excitement. It is most common between six months and two years of age.

SYMPTOMS.—The attack begins suddenly, more often at night, with dyspnœa and crowing inspiration, which in a few minutes may pass off, and after crying the child may soon fall asleep again. In other cases, the severity and duration are more marked, and all respiratory sounds may actually cease for a short time, owing to complete closure of the glottis, and death from asphyxia is imminent. But at this crisis the laryngeal spasm as a rule suddenly ceases, and the attack quickly subsides. Spasmodic flexion of the thumbs and great toes, or general convulsions, may be associated with the laryngeal phenomena. There is no pyrexia. Such attacks are very prone to recur; but with appropriate treatment, the ultimate prognosis is usually favourable. The possibility of death from general convulsion in the course of an attack must always be kept in view, though it is not a frequent occurrence.

Laryngismus differs from spasmodic laryngitis, by its occurrence at an earlier age, the absence of fever and cough, the convulsive symptoms with which it is associated, and the complete cessation of all symptoms between the paroxysms. Laryngismus must not be confounded with the curious crowing on deep inspiration, which young infants sometimes exhibit, and which results from abnormal

incurvation of the epiglottis. In none of the cases that the writer has seen, has it interfered with the child's well-being.

TREATMENT.—*During the attack* the child should be placed in a warm bath and cold water dashed on the body. This should be followed up by the injection *per rectum* of potassium bromide gr. iij.—v., with a grain or two of chloral, in solution, to be repeated as required. In bad cases inhalation of chloroform may be resorted to.

After the attack the treatment is mainly that of rickets, dyspepsia, and constipation, which are so frequently responsible for the disorder. No medicine is more useful for temporary relief of the convulsive tendency than bromide of potassium.

LARYNGEAL PARALYSIS.

The commonest form of laryngeal paralysis is a *bilateral paralysis of the adductors*. It is a purely functional affection, probably of central nervous origin. It is usually a manifestation of hysteria, in which case it is apt to be associated with anæsthesia of the larynx; but it is not an uncommon result of moderate laryngeal catarrh in highly neurasthenic subjects. The voice is lost (*aphonia*), but the sounds of coughing, sneezing, and sudden uncontrolled laughter are unaffected. *Unilateral paralysis of the adductors* is infinitely rare. The laryngoscope shows that, on attempting to speak, the vocal cords either do not move at all, or only approach each other imperfectly. The prognosis is favourable.

Paralysis of the abductors is generally *unilateral*, as the result of pressure upon the vagus or recurrent laryngeal nerve by thoracic aneurism, mediastinal tumours, enlarged cervical glands, or goitre. More rarely, abductor paralysis depends upon bulbar paralysis or other nervous lesions of a degenerative nature, in which cases the paralysis

may be *bilateral*. Though the recurrent laryngeal nerve supplies *all* the intrinsic muscles of the larynx, it is a noteworthy fact that the paralysis is generally incomplete, and only affects abduction as a rule. So long as only one cord is affected, respiration and phonation may be normal, and the condition remains undetected, apart from laryngoscopic examination, showing that the affected cord is not drawn outwards on inspiration. Some dysphonia, however, is not uncommon, and when both cords are affected, stridor and serious dyspnoea occur.

The *treatment* is for the most part that of the affection to which the paralysis is due. Paralysis of the adductors due to hysteria may be often cured by faradisation—one pole being placed at the back of the neck, while the other is either introduced into the larynx, or applied to it externally. Paralysis of the abductors may call for tracheotomy.

SUB-SECTION III.

DISEASES OF THE LUNGS.

BRONCHITIS.

Bronchitis is a catarrhal inflammation of the mucous membrane lining the bronchial tubes, which may on the one hand be limited to the trachea and larger bronchi (*simple bronchitis*); or on the other hand, may extend to their ultimate ramifications (*capillary bronchitis*). With respect to its course, it may either be *acute* or *chronic*.

ACUTE BRONCHITIS.

This condition usually occurs as the result of “catching cold.” In certain cases it appears to be distinctly infective; and no doubt the affection is generally of a microbic nature. It is often associated with specific fevers, such as measles,

typhoid fever, etc. Previous attacks, early or advanced age, sedentary indoor habits, and changeable damp climate are common predisposing causes.

MORBID ANATOMY.—As in all catarrhs, we meet with congestion, swelling, and desquamation of the mucous membrane, attended with mucoid or muco-purulent secretion. While in the larger tubes the swollen mucous membrane does not appreciably lessen the calibre, in the capillary tubes occlusion readily occurs, and pulmonary collapse is therefore a common result.

Also, since the terminal bronchi merge into the air-cells, the catarrhal process is very apt to extend from the former into the latter, and give rise to catarrhal pneumonia.

SYMPTOMS.—1. *Simple bronchitis*, confined to the larger tubes, is usually ushered in with chilliness, febrile disturbance, and general malaise. A sense of tightness about the chest and sub-sternal soreness soon follow. The respiration is increased slightly in frequency, and cough is uniformly present. At first the cough is dry and irritable, but afterwards becomes easier and looser. Expectoration is at first scanty and viscid, but soon becomes more abundant, white, and frothy, and later on, yellowish or muco-purulent. Duration is uncertain, but recovery is the rule. At the same time, from frequent recurrence, it may become chronic.

2. *Capillary bronchitis* may be either primary or supervene on the milder form just described. It is most common in children, aged persons, and those in feeble health. In children the onset is usually sudden, attended with rapid rise of temperature. The child is hot and restless. The breathing is rapid—out of proportion to the pulse; the *alæ nasi* are in active movement; coughing is incessant, but gives no relief. In unfavourable cases the breathing becomes more difficult and the complexion more dusky; the expression is anxious, and the restlessness increases. At

this stage broncho-pneumonia or collapse of lung may supervene, with delirium which merges into a fatal coma in the course of a few days or less. On the other hand, the cough may become looser, the breathing easier, the pulse slower and stronger, and recovery may take place.

In old people, profound exhaustion is the prominent feature. There may be no fever, little cough, and only moderate dyspnœa. Expectoration may be easy. But the weakness increases, the patient becoming drowsy and depressed, and though recovery is still possible, death from asthenia is quite common. Apart from careful physical examination of the chest, there may be little to account for it. Sometimes the case drifts into the chronic form.

PHYSICAL SIGNS.—In the milder forms of simple bronchitis physical signs may be absent, or limited to occasional wheezing or moist adventitious sounds. In capillary bronchitis the chest as a whole may be somewhat enlarged, and in children the lower ribs are often drawn in with each inspiration, from obstruction to the entry of air. The percussion-note is not much altered, but may be impaired over the posterior bases. The breath-sounds vary in intensity, and are frequently masked by abundant noisy rhonchi of sonoro-sibilant, mucous, or sub-mucous quality, altered by coughing. In bad cases, the breath-sounds may be almost suppressed, and masked by abundant moist sounds.

The *diagnosis* of simple bronchitis offers no difficulty, although care should be taken not to confound cases of primary bronchitis with specific fevers in which the catarrh is symptomatic, as for instance in typhoid fever. Capillary bronchitis, on the other hand, may be very difficult to distinguish from acute catarrhal pneumonia and acute miliary tuberculosis of the lungs, from the fact that it is usually associated with each of these affections. With regard to catarrhal pneumonia, the distinction is of little practical importance; but the more serious prognostic

significance of miliary tuberculosis places it in a different category. In *acute tuberculosis* the family and personal history, the violence and irregularity of the fever, the rapid collapse, and the extreme dyspnoea, out of all proportion to the physical signs, should help to indicate the nature of the case.

As to *prognosis*, early childhood, advanced age, previous organic disease of the lungs or heart, and the occurrence of complications, are especially of unfavourable import.

TREATMENT.—*Simple Bronchitis.*—The patient should be confined within doors, or if a bad case to his bed, at a warm equable temperature. It is most important to get the skin to act freely by hot air, vapour, or water baths, by warm diluent drinks, and by diaphoretic salines, which may be combined with a small dose of Dover's powder at night if there is much restlessness. Calomel followed by a saline purge is generally desirable at the outset. The diet should be light and nourishing, but alcohol is rarely necessary. So long as the cough is painful and ineffectual, relaxing expectorants such as ipecacuanha and antimonial wine are indicated. The inhalation of steam is soothing, and the addition of tr. benzoin. co. may be helpful. Hot fomentations to the chest may afford much relief. When the temperature falls, and the cough becomes loose, fomentations may be dispensed with, in favour of simple cotton-wool; while, medicinally, such drugs as carbonate or chloride of ammonium, in combination with alkalies and infusion of bark, are indicated.

In the *capillary bronchitis of young children* we have not only to deal with feebler powers of resistance, but with a greater liability to pulmonary collapse and broncho-pneumonia. For the removal of secretion nothing is more useful than an early emetic. Inhalations of steam are very beneficial. As to drugs, belladonna in doses of $\text{m i.}-\text{v.}$ of the tincture, every three or four hours, deserves

special mention. It is often useful, when collapse of lung threatens, to put the child into a hot bath, and dash cold water over the chest. In other respects, the general principles for the treatment of simple bronchitis apply.

In aged persons, we have to rely chiefly upon good nutritious food, alcoholic stimulants, inhalations of oxygen, and hypodermic injections of strychnia.

Quinine and strychnia are useful tonics in convalescence from acute bronchitis.

CHRONIC BRONCHITIS.

This is a very common disease, its frequency increasing with age. It may be chronic from the outset, especially when associated with cardiac, renal, or gouty affections; but it frequently occurs as the sequel of acute or sub-acute attacks. Chronic lung affections of other kinds, and the introduction of irritating vapours or particles into the tubes are also common causes. Chronic bronchitis is generally associated with emphysema, by which the troubles of bronchitic patients are greatly increased.

MORBID ANATOMY.—The mucous membrane is dark-coloured, thickened, and its vessels are dilated. The epithelial lining—ciliated and otherwise—is largely shed, and is replaced by irregularly cylindrical and ovoid cells. The bronchial walls gradually become thickened and indurated, the tube-cavities may be narrowed or dilated, and usually contain muco-purulent matter or frothy mucus. More or less emphysema is generally present.

SYMPTOMS.—The following are the most important varieties of chronic bronchitis :

1. "*Winter cough.*"—This is a common affection of elderly persons, especially those in feeble health, those exposed to bad weather, and those who take alcohol in excess. The cough is at first most troublesome in the mornings, and is accompanied with frothy, mucoid expectoration. Later

on, it is more frequent, and the sputa become more abundant and muco-purulent. There is more or less dyspnœa, in proportion to the amount of emphysema. At first the symptoms are relatively slight, and are confined to the winter months, disappearing in the summer. Each year, however, the attacks tend to be more severe, and of longer duration, till the patient is never quite free. Dyspnœa is now present, even when the patient is at rest; and attacks of an asthmatic nature are apt to occur, especially at night. In the final stages there is marked emaciation, and general ill-health. The dyspnœa may become so bad that the patient can only breathe when propped up in bed, or sitting in a chair. The complexion becomes dusky, the veins engorged, together with all the symptoms of dilatation of the right side of the heart, until death occurs from slow asphyxia or heart failure.

2. *Dry catarrh* is especially characterised by very scanty expectoration. The cough is violent and paroxysmal, the breathing is oppressed and wheezing, with a feeling of tightness on the chest. This form is most common in gouty subjects.

3. *Bronchorrhœa* generally occurs in old people, and is characterised by copious expectoration of a thin watery fluid, but sometimes ropy and glutinous, and occasionally foetid. The cough and dyspnœa are marked. This variety is especially apt to be associated with bronchial dilatation (*bronchiectasis*).

The *physical signs* of chronic bronchitis, as distinguished from those due to associated emphysema and bronchial dilatation, are limited to sibilant, sonorous, mucous, or bubbling râles, more or less generally distributed over the chest, but usually more abundant towards the base.

Chronic bronchitis becomes a serious affection from its leading so frequently to emphysema and bronchial dilatation, for when these secondary changes are marked, re-

covery—in the strict sense—is out of the question ; and when nutrition begins to fail, and dilatation of the right side of the heart from obstruction of the pulmonary circulation supervenes, the end is usually not far off.

In the course of chronic bronchitis we occasionally meet with peculiar decomposition of certain areas of the bronchial mucous membrane, giving a horribly foetid and gangrenous odour to the breath and sputa. It usually begins rather abruptly ; and, after a variable time, may pass off without permanent injury. Sometimes, however, it sets up a serious general septic infection ; and, in rare cases, it may lead to gangrene of the lung. It is very commonly associated with bronchial dilatation.

The *treatment* varies very much according to circumstances. Exposure to cold and damp should be avoided. Warm clothing is essential, but it should not be overdone, as the body thus becomes unduly sensitive to cold. To prevent this, tepid or cold morning baths, and plenty of exercise in the fresh air, should be urged during the summer-time, provided the general health and strength permit. Strict attention should be paid to the hygienic surroundings of the patient. As to medicines, a course of cod-liver oil—with or without malt extract—may be very beneficial during the winter months. For the cough, alkalies are of great service ; and warm milk with alkaline mineral water may be taken in the early morning with advantage. Inhalations of steam, to which tr. benzoin. co. may be added, are also useful for the same purpose.

When expectoration is difficult, salines and relaxing expectorants are indicated ; but otherwise tonic and stimulating remedies are to be preferred, such as ammonia and bark, in association with chloride of ammonium or iodide of potassium. Stimulating liniments to the chest are also of considerable service. In chronic cases with abundant secretion, turpentine, terebene, tar, or creosote deserve

trial. They may be taken internally, or be administered by inhalation—either dry, or in combination with steam.

The diet should be plain, easily digestible, and moderate in amount. Alcohol is frequently required, especially in old people, but it should be given in response to definite indication, and not as a matter of routine.

BRONCHIAL DILATATION.

Dilatation of the bronchi, or bronchiectasis, though a purely secondary affection, deserves separate mention both on diagnostic and prognostic grounds. No distensile pressure likely to occur in the course of disease is adequate by itself to cause permanent dilatation of the bronchi. Diminished resistance of the bronchial wall as the result of pathological change is a necessary antecedent, and generally arises from chronic bronchitis, broncho-pneumonia, or some general infection such as influenza or measles. Given diminished resistance, the walls yield under the distensile strain of severe and repeated coughing, or increased negative pressure from collapse or fibroid shrinkage of the lung. Dilatation may affect considerable lengths of the bronchial tubes (*cylindrical dilatation*), or be restricted to localised parts (*saccular dilatation*), but they are often associated. The former chiefly affect the larger and medium bronchi; while the latter generally involve the smaller and terminal bronchi. Saccular dilatations are more important clinically, because they are liable to be mistaken for cavities produced by tuberculous ulceration, and because they so often lead to retention and decomposition of secretion. In size and number they vary widely. As a rule, they do not exceed $\frac{1}{2}$ inch in diameter, and are often much smaller, and for the most part lie fairly near the surface of the lung. Their number is roughly inverse

to their size. They are lined by a smooth or granular membrane, continuous with the mucous membrane of the tubes with which they communicate. Dilatations of the bronchi are more common in the lower lobes of the lungs, corresponding to the distribution of the morbid process to which they are secondary. In the later stages, bronchiectasis is almost always associated with marked fibrosis of the lung, whether previously present or not.

The *diagnosis* of bronchial dilatation rests chiefly upon a history of chronic bronchitis or broncho-pneumonia; antecedent pleurisy, the paroxysmal character of the cough, the abundance and frequent fœtor of the sputa, and (in saccular forms) the physical signs of cavities which are more often in the middle or lower lobes of the lungs, and the very slow progress of the disease. The difficulty of diagnosis from phthisis is increased by the malnutrition, clubbed finger ends, fever, and occasional hæmoptysis which sometimes accompany bronchiectasis. Careful examination, however, of the sputa will usually suffice to clear up the nature of the case, except where (as sometimes happens) both conditions are present.

The treatment is purely palliative. Relief is best afforded by the internal administration of terebene or turpentine, together with antiseptic inhalations of carbolic acid or eucalyptol.

PLASTIC BRONCHITIS.

This is a rare form of bronchitis, characterised by the expulsion of fibrinous moulds of the small bronchi, which are often branched. These bronchial casts must not be confounded with those resulting from the extension of diphtheria into the air passages, nor with the blood casts which sometimes occur in hæmoptysis. Nothing is known

of the conditions on which it depends, though analogy with other membranous inflammations would suggest the probability of microbic origin. It is sometimes associated with tuberculosis. The casts are of a whitish colour, and of a fibrinous nature. The affection is more common in males of early middle life.

Though acute cases are recorded, the affection is generally chronic, and is characterised by attacks at variable intervals of dyspnœa and cough, attended by the characteristic sputa. The onset of an attack may be sudden; but more often it is preceded for a while by the signs of ordinary bronchial catarrh. The severity and duration of the dyspnœa depends on the size and number of casts and their rate of formation, and great relief is given by their expulsion. The sputa, apart from the casts, are abundant and fluid, and sometimes streaked with blood. Slight fever may be noted, but more often the general health is little affected. The affection may be easily mistaken for asthma, unless the sputa are carefully examined. When uncomplicated it is rarely fatal, but a liability to recurrence may continue for an indefinite period. There are no special physical signs, apart from associated bronchitis.

As to treatment, nothing is known which will control the exudation.

ASTHMA.

Asthma may be briefly defined as a disease characterised by *paroxysmal dyspnœa*. An *asthmatic paroxysm* may be preceded for a variable time by a sense of oppression and constriction of the chest, with wheezy respiration, or in some cases by the abundant discharge of pale watery urine. On the other hand, it may come on without any warning.

In either case the actual attack occurs most commonly at night. The patient awakes scarcely able to breathe, adopts the sitting or erect posture with the shoulders raised and elbows fixed, the head thrown back, and the mouth open, and all the extraordinary muscles of respiration are brought into play. The face wears an aspect of terror; the eyes are widely opened; the skin is pale at first, but later becomes dusky, and often bedewed by sweat; the extremities are cold; and the pulse is small and quick. The breathing, however, is not hurried, and may be slowed; and while inspiration is short, expiration is inordinately prolonged.

The resonance of the chest on percussion is increased, vesicular breath-sounds are weak, expiration is prolonged, and dry sibilant râles are heard. These physical signs very commonly exhibit a patchy distribution.

Towards the end of an attack, cough occurs with expectoration of small, firm, non-aërated pellets of mucus, some of which may have a spiral structure. The *duration* varies greatly. The paroxysm may pass off in the course of an hour or two, but more often it lasts for much longer, and sometimes continues on and off for days. Cough, wheezing, and expectoration often persist for some days afterwards. Recurrence is the rule, and is apt to assume a more or less periodic character. In favourable cases, the intervals generally become longer, until the attacks cease. On the other hand, in a large number of cases, they persistently recur at variable intervals, and though rarely fatal, frequently spoil a patient's life. In old age the paroxysmal feature disappears, and is gradually replaced by the permanent dyspnoea of emphysema. Bronchitis and emphysema are common accompaniments.

ETIOLOGY AND PATHOLOGY.—It has been often taught that, while some cases of asthma are *symptomatic* or secondary to other morbid conditions, such as pulmonary,

cardiac, or renal disease, others occur independently of such causes, and are therefore termed *primary* or *nervous*. Such a distinction seems to introduce an unnecessary confusion into the subject. Asthma is a disease with definite characters of its own, and should not be confused with symptomatic dyspnoea of a paroxysmal nature. The most important facts relating to its pathology, in the writer's opinion, appear to be these. In the first place, the suddenness both in the appearance and disappearance of the attacks unquestionably suggests a nervous origin. According to this view, asthma is a neurosis, affecting the nervous centres in the medulla which preside over the respiratory mechanism, and the blood supply of the lungs. A special excitability of these centres, and a liability to disturbance, either inherited or acquired, must be assumed as a primary factor in the etiology of asthma under *all* circumstances. Authorities agree in attributing the immediate cause of the attack to obstruction in the finer bronchi. Two conditions are put forward as responsible for the obstruction—spasm of the bronchial muscular coat, and engorgement with swelling of the respiratory mucous tract through vaso-motor agency. As to their respective importance, there is still difference of opinion, but the weight of evidence is in favour of the theory of spasm, not only of the bronchial muscular fibres, but of the inspiratory muscles generally. This paroxysmal excitement of the respiratory nervous mechanism may in certain cases be of central origin, as in asthmatic attacks brought on by fright, or violent emotion of any kind. More often, however, the attack is induced by peripheral irritation, such as inhalation of dusty particles, odours of certain kinds, bronchial catarrh, nasal polypi, post-nasal adenoids, dyspeptic disorder, constipation, or worms. Climatic conditions again have often an important, though elusive influence, a patient having frequent attacks in one place,

while exempt in another. The special liability to attacks during sleep may be probably explained by increase of carbon dioxide in the blood at this time, and by confinement—as too often happens—in close and impure air.

The *diagnosis* rests upon the paroxysmal and usually sudden nature of the attacks, with more or less complete recovery in the intervals between them.

TREATMENT.—*During the paroxysm.* Remove tight clothing and give fresh air.

As to medicines, unless contra-indicated by tender age or abundant bronchial secretion, nothing can be trusted better to cut short or relieve the distress than the subcutaneous injection of gr. $\frac{1}{8}$ – $\frac{1}{6}$ of morphia. When morphia is undesirable for any reason, chloral hydrate deserves trial. Of course remedies of this class, however successful, must not be indefinitely repeated. Among other measures, the inhalation of nitrite of amyl, or the fumes of burning stramonium or nitre-paper deserve mention. In cases dependent upon nasal or faucial irritation, the use of a cocaine spray will sometimes cut short an attack.

During the interval.—A painstaking investigation of the probable cause with a view to its correction or removal is of the first importance. Thus much may generally be done by regulation of the diet on simple lines, with avoidance of alcohol, tea, coffee, and tobacco. An occasional dose of calomel at night, followed by a saline aperient next morning, is desirable. Of drugs, the most generally useful are salicylate of soda, potassium iodide, and arsenic. Climatic treatment demands careful consideration; but it conforms to no known law.

PULMONARY CONGESTION AND OEDEMA.

Apart from collateral congestion of the lung, as for instance in a case of large pleural effusion on the opposite

side, and apart also from congestion due to suction-action on the pulmonary vessels from inspiratory obstruction of any kind, it may be said that active congestion of the lung always represents either an early stage, or a minor degree of inflammation.

Mechanical congestion of the lungs is a common result of valvular disease of the heart. *Passive* congestion depends upon enfeeblement of the heart's action, and is a common complication of fevers and other conditions characterised by great debility and prostration. Under the influence of gravitation, it especially affects the most dependent parts of the lung (*hypostatic congestion*). When congestion is severe or long-continued, other changes are apt to follow—*e.g.* pneumonia, hæmorrhage, and œdema. Congestive œdema of the lungs is favoured by conditions which impair the quality of the blood or tissues, such for instance as in renal dropsy.

MORBID ANATOMY.—In mechanical congestion, the lungs are large, engorged with blood, of dark red colour. In hypostatic congestion, the bases and posterior parts of the lungs are of dark red colour, engorged with blood, and in a more or less infiltrated and airless condition (*spleenization*). In œdema, the lungs are heavy, swollen, pit on pressure, and yield on section a large quantity of watery serum.

SYMPTOMS.—These vary according to the severity and duration of the congestion, and the amount of œdema. More or less cough and dyspnœa are always met with, and sometimes in association with oppression at the chest, and duskiuess or lividity of the cutaneous surface. The expectoration is mucoid, frothy, and sometimes bloody in mechanical congestion, while it is profuse and watery in œdema dependent upon general dropsy.

The physical signs are not characteristic, and are generally limited to weak and harsh breath-sounds, with mucous or thin bubbling râles, which are more abundant over the

bases of the lung, and for the most part symmetrically distributed. In œdema and hypostatic congestion, there may also be some dullness of the percussion note over the bases posteriorly.

The *prognosis* and *treatment* necessarily vary according to the condition to which the congestion is due.

PULMONARY HÆMORRHAGE—HÆMOPTYSIS.

Hæmorrhages into the air-passages, attended with spitting of blood or hæmoptysis, may be due to a variety of causes :—(a) ulcerative affections of the larynx or trachea ; (b) congestion, especially that occurring in connection with valvular disease of the heart ; (c) various diseases of the lung, particularly in pneumonia, tuberculosis, and cancer, and more rarely in bronchiectasis, abscess, or gangrene ; (d) perforation of an aneurism into the air-tubes ; (e) certain hæmorrhagic affections, such as purpura, scurvy, hæmophilia, and the malignant forms of some specific fevers, and (f) vascular degeneration, such as occur in gouty persons, or in old age. To this list may be added the hæmorrhage which occurs from hæmorrhagic infarction of the lung.

Hæmorrhagic infarction is generally the result of embolic plugging of a branch of the pulmonary artery by a clot from the right heart or a systemic vein ; but it is often difficult to distinguish true infarction from extravasation of blood into the lung, due to simple rupture of a pulmonary vessel from mechanical engorgement. Infarcts are wedge-shaped when they occur at the periphery of the lung, the base coinciding with the pleural surface ; but in the deeper parts of the lung, they are irregularly rounded. They vary much in size, are firm and solid, and dark red on section. The blood lies in the air-cells and air-tubes, and there is little or no breaking down of lung tissue. Necrosis is rare, and in

course of time the blood is reabsorbed, and the part recovers, or shrinks into a brown indurated nodule.

SYMPTOMS.—*Hæmoptysis* is the characteristic symptom of pulmonary hæmorrhage. It varies from a mere staining of the sputa or the presence of a few streaks, to large quantities of pure blood. When the amount is small it is expelled by spitting; when larger it escapes in gulps, or with efforts scarcely distinguishable from vomiting. The blood is usually bright red, alkaline, and frothy; but when its discharge is delayed the colour is darkened; and when the bleeding is profuse, it is non-aërated. Attacks of hæmoptysis are prone to recur; and *for some days after each attack, the sputa are always more or less tinged*. Apart from aneurismal rupture, a single attack is rarely fatal, and the ultimate prognosis depends almost entirely on the cause of the bleeding. A careful inspection and the dark colour of the blood will usually suffice to distinguish bleeding from the nose or mouth; while laryngoscopic examination and the presence of laryngeal symptoms will generally enable us to detect hæmorrhage from this source. Hæmoptysis will be contrasted with hæmatemesis when describing the latter. The special points to be noted are the ejection by spitting, the bright red colour, the alkaline reaction, the frothy appearance, the tinging of the sputa afterwards, and the usual co-existence of disease of the lungs, heart, or mediastinum. On physical examination of the chest, moist sounds may be heard, and in some cases an area of dulness may be defined over the seat of hæmorrhage.

Even when fairly assured that the bleeding is from the air-passages of the lung, it is often very difficult to refer it to its exact cause. Careful examination of the chest is of the first importance, and the results thus obtained must be considered together with the age of the patient, the history of the case, and the general condition. Hæmoptysis is more often associated with phthisis than with any

other affection, and not infrequently constitutes the first obvious indication of this disease; though on inquiry it will almost always be found to have been preceded by progressive loss of flesh. Hæmoptysis and dyspnœa suddenly occurring in connection with valvular disease of the heart, or thrombosis of a peripheral vein, point to pulmonary infarction. The diagnosis of the rarer forms of hæmoptysis caused by cancer, aneurism, etc., depends entirely on the general evidence of the existence of these conditions.

The general principles of *treatment* vary according to the condition giving rise to the bleeding. In the hæmoptysis of phthisis, for instance, rest in bed in the recumbent posture in a cool room, the avoidance of alcohol and hot food or drink, and the control of coughing by sucking ice or by a sedative linctus, are indicated. The hypodermic injection of morphia is often most beneficial as a general calmative. The value of astringent drugs is questionable, but chloride of calcium in gr. x. doses, 3 or 4 times a day, appears to be worthy of trial. In hæmorrhage dependent upon valvular disease of the heart, on the other hand, this treatment would be worse than useless; and such measures as purgation by calomel followed by saline aperients, leeches, or dry-cupping over the chest, and the administration of digitalis are indicated.

ACUTE PNEUMONIA.

The term "pneumonia" is often used in a general sense to indicate any inflammatory affection involving the parenchyma of the lung. But it is here restricted to an acute general specific infection, with pulmonary inflammation as a local manifestation. The organism on which it depends is known as the *pneumococcus* of Fraenkel, con-

sisting of oval cocci, generally arranged in pairs. In fresh sputa they are surrounded by a distinct capsule, which, however, is not evident in artificial cultures. Although a majority of cases yield a pure culture of pneumococci, secondary infections are quite common, and this may explain certain clinical varieties of the disease. It is probable that, under typical conditions, the general infection precedes the local changes in the lung, the latter being in fact secondary to the former. This view is in no way incompatible with the assumption that the local pneumococcal lesion contributes, when established, to the general infection. Other inflammations may sometimes occur as the result of the general infection, such as pleurisy, meningitis, endo- or pericarditis, or suppurations in different parts of the body, with or without associated pneumonia—all standing in precisely the same relation to the primary infection. Invasion is followed, under ordinary circumstances, by a protective general reaction, indicated by fever, leucocytosis, and the formation of anti-bodies; which, by the end of about a week, have become capable of overcoming the toxæmia. Whereupon, the temperature falls by *crisis*, the leucocytosis subsides; and henceforward, the pulmonary lesion behaves as a purely local inflammation which gradually resolves. On the other hand, in highly virulent infections, and in feeble subjects, the general reaction may be slight and inadequate, the toxæmia is not controlled, and a fatal result is the rule. The pneumococcus is very generally present in the secretions of the throat in healthy persons, a fact which largely explains the great liability to pneumonia when the bodily resistance is lowered from any cause.

Alcoholism, cold, inanition, and senility, and, in short, all conditions attended with serious depression of vitality, rank as predisposing causes. Pneumonia occurs at all ages; more commonly among males than females. It

prevails chiefly in the winter and spring seasons of the year; and one attack confers no protection against its repetition.

MORBID ANATOMY.—Three stages are generally described. (1) The first *stage of engorgement* is characterised by active congestion of the lung. (2) In the *stage of exudation or red hepatisation* the inflamed lung is of dark red colour, enlarged, solid, inelastic, non-crepitant, and heavy. It breaks down readily on pressure, and sinks in water. On *section*, the surface is dark red, mottled with grey, and presents a peculiar *granular* appearance, due to protrusion of the fibrinous plugs from the divided air-cells. Microscopically, the vesicles and terminal bronchioles are filled with a fibrinous exudation, enclosing both red and white blood-cells, together with pneumococci. Up to this point there is little or no alteration in the alveolar walls. (3) The *stage of grey hepatisation* is characterised by a gradual change in colour from dark red to grey or yellowish-white, beginning in the older and deeper parts of the inflamed area, and extending peripherally. The weight, density, and friability of the lung are increased, but the granular appearance on section is less marked. Microscopically, the air-cells and interstitial tissue are found to be crowded with leucocytes, while the red corpuscles and fibrin begin to liquefy and disappear.

If the patient survives thus far, the usual termination is *recovery* by liquefaction of the exudation, due to enzymes set free by the leucocytes, and its removal by expectoration and absorption. In rare cases, the lung tissue breaks down into pus, with the formation of an *abscess*. Still more rarely, it ends in *gangrene* of the affected part.

One lung is more frequently attacked than both, the right than the left, and the lower lobe much more often than the upper. The unconsolidated parts are, as a rule, more or less emphysematous, with hypostatic congestion at the

base. The pleura covering the affected part is generally inflamed.

SYMPTOMS.—The attack usually sets in abruptly with chilliness or rigor, followed by a sharp rise in temperature; but it is not uncommon for the onset to be preceded by indications of ill-health for an uncertain period. In children, the onset is often marked by vomiting, and sometimes by convulsion. Three important local symptoms now occur. (1) *Pain* in the affected side—sharp, stabbing, aggravated by cough, deep inspiration, or pressure. (2) *Dyspnœa*—characterised by extremely frequent shallow breathing, out of all proportion to the rate of the pulse (often 1 to 2·5, or less). It usually ranges from 30 to 60 per minute; and is due, partly to the fever, partly to malaëration of the blood, and partly to the pain. The *alæ nasi* are in active motion. (3) *Cough* is frequent, hacking, and restrained on account of the pain. It is dry at first, but is soon attended with *viscid, tenacious, rust-coloured expectoration*. Sometimes there is genuine hæmoptysis, but it is not necessarily of serious significance. Expectoration does not occur in young children; and it is often absent in aged, feeble, or alcoholic subjects.

GENERAL SYMPTOMS.—The *fever* attains its maximum on the second or third day, usually ranging from 103° to 105°; and, but for small daily variations, this temperature is usually maintained until the *crisis*. A high temperature *at the onset* is not by itself necessarily serious. Marked temporary remissions are sometimes observed, particularly on the day preceding the crisis, but are not attended by any relief of the constitutional disturbance. The *skin* is excessively hot, dry, and pungent. The *pulse* is frequent, from 80 to 120 per minute, usually large and full at the outset; but it afterwards becomes small and weak, partly as the result of imperfect filling of the arteries from obstruction to the pulmonary circulation, and partly

through cardiac feebleness. The *cheeks* are flushed (sometimes only on the affected side), and *herpes* is often present on the lips. Prostration, feebleness, headache, sleeplessness and slight nocturnal delirium, are common *nervous symptoms*. The *tongue* is coated and dry; *thirst* is increased; the *appetite* is lost; and the *bowels* are usually costive. The *urine* is of the febrile type; chlorides are deficient or absent, and it may contain a little albumen.

The above symptoms continue for five to ten days, when, in favourable cases, they quickly subside, the skin becomes moist, and the temperature rapidly falls to normal, constituting what is called defervescence by *crisis*. The sputa, which have already lost their rusty colour, now become more abundant, yellowish, and muco-purulent. Defervescence is sometimes gradual. When the disease ends fatally, death usually occurs on the fifth day, or a day or two later, either from cardiac failure or toxic adynamia. More rarely, it occurs from obstruction to breathing, hyperpyrexia, collapse after the crisis, or from complications. Among the latter, pleurisy and pericarditis are the most important.

PHYSICAL SIGNS.—Fine crepitation at the end of deep inspiration may be the earliest sign, but it is quite temporary, and may easily be missed. Movement is often slightly diminished on the affected side. As a rule there is no alteration in the percussion-note during the first twenty-four hours; and, when the pneumonic area is deep seated, it may be delayed still later. Otherwise, as soon as consolidation has occurred the note becomes dull. Not infrequently, however, in early stages, the note has a somewhat tympanitic quality. Above the dull area the same kind of resonance (Skodaic) may be observed in many cases. At first the breath-sounds are simply weak. As consolidation advances, the vesicular quality is rapidly lost, and replaced by breath-sounds of a dry, high-pitched,

bronchial type, with increased vocal resonance (bronchophony). With the commencement of resolution a moist crepitation (*crepitation redux*) develops, increasing as resolution proceeds. There is no change in the physical signs corresponding to the crisis; and they only slowly subside over a period of variable duration.

VARIETIES.—The disease may be *latent* in respect of both symptoms and physical signs. In aged and alcoholic subjects there may be few or no characteristic symptoms; and, apart from physical examination, diagnosis may be impossible. On the other hand, in small deep-seated lesions, there may be no physical signs. Cases also occur in which the real affection is *masked* by the prominence of nervous disturbance. A *septic* or *typhoid* type is sometimes met with, in which the pulmonary symptoms are less pronounced, and often mingled with those of bronchial catarrh, accompanied with marked nervous disturbance, a brown dry tongue, irregular fever, and (in event of recovery) slow defervescence. In rare cases when the exudation involves the bronchioles as well as the air-cells, the physical signs closely resemble those of a pleural effusion (*massive pneumonia*). Lastly, pneumonia may be of an asthenic type, *creeping* about from one part of the lung to another.

The *diagnosis* mainly turns on the sudden onset, the high fever, the altered pulse-respiration ratio, the rusty viscid sputa, and physical signs. It must not be forgotten that, in young children and in drunkards, the disease is often masked by prominent nervous symptoms.

As to *prognosis*—old age, infancy, drunken habits, the affection of both lungs, and extreme nervous disturbance are of unfavourable import. Secondary pneumonia (*cateris paribus*) is more serious than primary pneumonia. A leucocytosis under 25,000 is unfavourable.

TREATMENT.—Since little or nothing can be done either,

to subdue the inflammation or to shorten the course of the disease, the chief objects of treatment are to sustain the strength of the patient, and relieve symptoms as they arise. Rest in bed is imperative, in a well-ventilated room, at an equable temperature, and the patient should be kept as cool as possible. The diet should consist of milk, eggs, beef essence, etc., and should be supplied according to the digestive capacity of the patient; but excessive feeding, especially with raw milk, is undesirable, as tending to flatulent distension. Watery drinks should be freely allowed. Alcohol is always necessary among the aged, also in most cases of secondary pneumonia, and whenever there are evidences of progressive debility and cardiac failure, or inability to take sufficient nourishment. When indicated, it should be given freely. At the onset a brisk calomel purge is desirable, which should be followed in ordinary cases by a saline, such as liq. ammon. acetatis and citrate of potash, or an effervescing quinine mixture, every four hours. All depressants should be avoided. Later on, the medicinal indication depends upon circumstances. In simple cases, nothing may be needed beyond the saline medication first described. But whenever the age, previous habits, and present condition of the patient make it probable that the disease will assume an asthenic type, alcohol and tonic remedies such as quinine, ammonia and bark, in combination with digitalis, or nux vomica, are urgently required. Symptoms of asthenia should not be waited for, but foreseen, as far as possible. No pains should be spared in keeping the mouth and nasal passages clean.

Severe pain and restlessness in the *early stage* may be advantageously treated by Dover's powder, or the hypodermic injection of morphia (except in children); but later on opium is rarely admissible. In milder cases, hot fomentations or the application of ice-bags usually suffice. For

sleeplessness it is well to trust as far as possible to nourishment, to a judicious allowance of alcohol, to measures for keeping the patient cool, to careful attention to all that makes for his comfort, and to absolute quietness of the sick-room. Delirium may best be met by cold spongings, tepid sheet-packs, ice to the head, and a bromide draught, or a full dose of chloralamide in bad cases. Hyperpyrexia demands the employment of the graduated bath. The tendency to cardiac failure should be combated by full doses of digitalis internally, hypodermic injection of strychnia, and inhalation of oxygen. If not postponed till too late, this combination is of the greatest value.

BRONCHO-PNEUMONIA.

Broncho, catarrhal, or lobular pneumonia is most common at the extremes of life. When it occurs in adults it is practically always of tuberculous origin. Any cause capable of inducing a capillary bronchitis may thus indirectly give rise to broncho-pneumonia. Its course is for the most part acute, but in tuberculous cases its progress may be slow.

Broncho-pneumonia is always a secondary affection in the sense that it depends on extension of catarrhal inflammation from the bronchioles into the air-vesicles of the lung. And it is of secondary nature also in the fact that it often occurs in the course of acute infectious fevers, especially of measles, whooping-cough, and diphtheria. It also arises secondarily to aspiration into the air-tubes of food-particles, foreign material of various kinds, or blood. In contrast to such cases, it would be correct to say that broncho-pneumonia in certain cases (though a minority) is primary, in so far as it occurs apart from associated general or other local disease. Broncho-pneumonia has no special bacteriology. It is merely that of the organisms

which happen for the time being to predominate in the air-passages, such as tubercle or diphtheria bacilli, pneumococci or streptococci, etc., variously combined, as the case may be.

The bronchial origin of broncho-pneumonia is impressed upon its *morbid anatomy*. Thus the distribution of the lesion is racemose, corresponding to the affected bronchi. Areas of consolidation are associated with areas of collapse, and often of emphysema, secondary to the bronchial inflammation, giving the lung-surface on section a mottled or variegated appearance. The exudate in the air-cells and terminal bronchi is largely mucoid (*not* fibrinous), containing desquamated and fatty epithelial cells and leucocytes. The bronchial walls are œdematous, and infiltrated with leucocytes. Large areas of lung may be involved by fusion of adjacent pneumonic lobules, but otherwise we do not meet with lobar distribution as in acute pneumonia.

In favourable cases, the contents of the alveoli liquefy, and are either expelled or absorbed; but sometimes the condition becomes chronic, and, being associated with interstitial inflammation, results in more or less induration of lung, with areas of caseous degeneration. This result is most common in connection with tuberculosis. In cases arising from the presence of food or septic particles, the process may end in suppuration or gangrene.

The symptoms vary considerably according to the conditions under which the disease arises, the age of the patient, and the extent of the lesion. In children, more often it occurs as the result of bronchial catarrh in the course of some infectious condition such as whooping-cough or measles. The onset is then relatively insidious. The temperature rises, breathing becomes more rapid, and the cough worse. Dyspnoea is a prominent feature; the cough is hard and painful, and there is little or no expectora-

tion, as is the rule with young children. If any secretion is coughed up, it is not stained as in acute pneumonia. The body-surface is dusky or actually livid, and often bathed in cold and clammy sweat. Restlessness is extreme, and there may be delirium. Anorexia and thirst are almost invariable, and vomiting and irregular diarrhœa are not uncommon. The urine is scanty and high coloured. In fatal cases, asphyxial symptoms supervene, and the child passes into a state of stupor. In favourable cases, after an uncertain duration which is rarely less than ten days, the symptoms improve, and the temperature falls gradually to normal. The physical signs are mainly those of capillary bronchitis; but there may be areas of impaired resonance, bronchial or blowing breath-sounds, metallic râles, and bronchophony.

In primary cases in young children, the onset is abrupt, and often ushered in by vomiting or nervous symptoms; the temperature rises higher, and tends to subside by crisis. The pulmonary symptoms are less relatively to the extent of the constitutional disturbance. In point of fact, the behaviour is closely similar to that of acute lobar pneumonia.

In old people, the most characteristic feature is the extremely insidious way in which it develops. Fever, cough, and dyspnœa may be absent and, apart from physical examination (which may also sometimes fail to reveal striking signs), there is little to attract attention, except a rapidly progressive asthenia, which often proves fatal.

The *diagnosis* from associated bronchial catarrh is often difficult, and is not of much practical importance. The recognition of tuberculous disease may also be very difficult, but it demands careful consideration. A tuberculous history, the presence of tuberculous mischief elsewhere—in glands or joints, the longer duration, rapid emaciation, profuse sweating, and apical distribution point to tubercle;

while the detection of tubercle bacilli in the sputa makes it certain.

TREATMENT.—No lowering treatment is admissible. Internally, quinine in full doses generally answers better than anything else; ammonia and bark, with or without expectorants, is also a useful combination. Good nourishment is essential, and alcohol is often requisite. Externally, hot and moist applications are recommended, as in acute pneumonia. During convalescence tonics are indicated.

INTERSTITIAL PNEUMONIA—PULMONARY FIBROSIS.

This condition is characterised by the formation of fibroid tissue, which tends to obliterate and destroy the normal vesicular tissue of the lung. It represents, in a large majority of cases, the result of chronic irritation which may arise from a great variety of causes. Many are purely of a local nature; and, of these, tuberculous foci are by far the most numerous and important. On the other hand, pulmonary fibrosis is often diffuse, involving large areas, or even the whole of the lung. Thus it may occur as the result of chronic bronchitis, especially that form which depends upon the persistent inhalation of dusty particles, as in millers, miners, or steel-grinders. In other cases, it appears to originate in a dry pleurisy, extending from the thickened adherent pleura in the form of fibrous bands which penetrate the lung in all directions. These should be distinguished from the fibroid changes which take place in a lung which is collapsed and bound down as the result of chronic pleurisy. Lastly, there are rare cases which occur as the sequel of acute pneumonia, syphilis.

The anatomical character and distribution of fibrosis vary according to its cause. When a large part or the whole of a lung is involved, it is usually confined to one side. The organ is shrunken, airless, and firm; while the bronchi are dilated. Hypertrophy or dilatation of the right heart is of common occurrence. The opposite lung is emphysematous. The local form calls for no special description.

The *symptoms* are difficult to dissociate from those of the affection on which the fibrosis depends. In well-marked cases we meet with dragging pains in the side, dyspnœa on exertion, incessant paroxysmal cough, and expectoration, the amount and character of which vary according to the condition of the bronchi. When the bronchi are dilated, the expectoration is abundant, puriform, and often foetid. Loss of flesh and strength, moderate irregular pyrexia, clubbing of the finger-ends, night-sweats, and sometimes slight hæmoptysis may occur, independently of tuberculosis. But the frequent association of pulmonary fibrosis and tubercle should always be kept in mind, and periodical examination of the sputa for tubercle bacilli is necessary in order to avoid mistakes. In the later stages of well-marked cases, dilatation of the right side of the heart may give rise to trouble.

The *physical signs* may be few and equivocal; but usually there is evidence of retraction of the chest-wall, diminished respiratory movement, and displacement of the heart to the affected side. The results of percussion and auscultation are apt to be very equivocal, owing to the varied conditions which may be present—consolidation, dilated bronchi, and emphysematous areas.

The *treatment* is the same as that for chronic bronchitis with dilated bronchi.

PULMONARY TUBERCULOSIS—PHTHISIS.

Pulmonary tuberculosis is a specific local disease of the lungs, caused by the growth and development within them of the tubercle bacillus. The old clinical term "phthisis" has come to be used in practically the same sense; and though by no means exempt from objection, it has at least the merit of convenience.

It is well known that the sputa of phthisical persons are for the most part crowded with tubercle bacilli. Such sputa, when dry, become incorporated with the dust of the air, and are inhaled with the breath. This is no doubt the usual source of tuberculous infection of the lungs. Less frequently it is due to direct inhalation of spray caused by coughing on the part of affected persons. The milk of tuberculous cows is also an undoubted source of danger, especially in early childhood; but it tends more often to set up some form of abdominal rather than pulmonary tuberculosis. Yet again, phthisis may be induced by bacilli brought to the lungs from tuberculous lesions elsewhere, by the blood-vessels or lymphatics. Anything which reduces the vital powers, whether inherited or acquired, may rank as a general predisposing cause; while among local predisposing causes, bronchitis and old pleurisy are of chief importance. No age is altogether exempt from the disease, though it is rare at the two extremes of life. The mortality is highest between 20 and 30 years of age. Up to 30 years we have mainly to do with inherited predisposition; but, after that age, mainly with acquired predisposing factors.

GENERAL MORBID ANATOMY.—The lesions of pulmonary tuberculosis are of three kinds—miliary, caseous, and fibroid. *Miliary lesions* of the lung have been already referred to as a result of general tubercular infection (p. 126); but they also occur as the result of local infection from pre-existing tuberculous lesions in the lungs.

Caseous lesions are due to the introduction of tubercle bacilli by inhalation, and their subsequent arrest in the terminal bronchi and air-cells, where they give rise to isolated patches of broncho-pneumonia and peri-bronchitis. Under the necrotic influence of toxins produced by the bacilli, the inflammatory products thus formed undergo caseous transformation. In acute cases, the cheesy matter softens, breaks down, and leads to more or less destruction of lung tissue, with the formation of cavities; while, in chronic cases, it becomes firm, dry, and encapsulated by fibrous tissue, or undergoes calcareous degeneration.

Fibroid lesions, unlike those of a miliary and caseous nature, have nothing specific in their mode of origin, and merely represent a reactive result of chronic persistent irritation. They are characterised partly by fibrous changes in the tubercles themselves, partly by fibrous peri-bronchitis, and partly by a hyperplasia of the interstitial connective tissue of the lung.

Any one of the above lesions may so notably predominate in a given case, as to justify the recognition of special types of pulmonary tuberculosis, such as miliary, caseous, or fibroid; but the common and ordinary form of phthisis (which forms the basis of the description which follows) embraces both caseous and fibroid lesions, with intercurrent local miliary outbreaks. These differences in type are not quite easy to explain, but they probably have mainly to do with the quantity and virulence of the infective material, and also with variations in the general and local powers of resistance on the part of the patient.

CHRONIC BRONCHO-PNEUMONIC OR FIBRO-CASEOUS PHTHISIS.

ANATOMICAL APPEARANCES.—The disease as a rule begins at the apex of one lung; and, while extending from this point, independent centres of disease appear in other parts,

especially the apex of the opposite lung, and the upper part of the lower lobe on the same side. The process is usually described as occurring in three stages—viz. *consolidation*, *softening*, and *excavation*. If, however, we bear in mind that it is often impossible to determine exactly at what stage a case has arrived, and that different stages are met with even in the same lung, it follows that such a classification is of very limited practical importance.

The disease generally begins by the formation of tubercles in the finer bronchi, in association with localised catarrhal inflammation. Thence the inflammation extends (1) to the alveoli, and (2) to the peri-bronchial connective tissue in the immediate neighbourhood, forming small patches of chronic catarrhal- or broncho-pneumonia. Of the nodules of consolidation thus formed, some undergo caseous degeneration; which, beginning in the centre of each, extends peripherally, till the whole is converted into a structureless cheesy mass. In course of time the caseous mass may be encapsulated with fibrous tissue, or perhaps becomes calcified, and further mischief may be thus temporarily or permanently arrested. More often liquefaction of the inflammatory products takes place, communications with neighbouring bronchi are opened up, more or less purulent matter escapes and is discharged by expectoration, and cavities are thus formed. The latter are at first small and round, but may afterwards become large and irregular by coalescence with others. The lining surface, though rough and uneven to begin with, may ultimately become smooth and well defined.

Accompanying these changes—in direct proportion to their slowness of development—there is an increased formation of connective tissue, which leads to fibrosis and subsequent shrinkage of the affected area of lung. More or less bronchitis, bronchial dilatation, and pleuritic adhesion, are constant accompaniments of tuberculous

ulceration. Fresh crops of tubercle may appear from time to time, as the result of local infection. The small *pulmonary vessels* also often exhibit inflammatory or tuberculous changes in their walls, either leading to their rupture and consequent escape of blood, or to the coagulation of their contents. Sometimes they are the seat of *aneurismal dilatations*, which may burst and give rise to very serious hæmorrhage.

SYMPTOMS.—The onset of the disease is gradual, and characterised by features which vary considerably in different cases. It may originate (1) with an ordinary “cold,” which leaves behind it an intractable cough, accompanied with expectoration; (2) insidiously, with dyspepsia, anæmia, general malnutrition, and wasting; (3) with an attack of pleurisy; (4) with chronic laryngitis, which ultimately turns out to be tuberculous; or (5) with an attack of hæmoptysis.

Of the symptoms which attend the course of the disease, some are attributable more or less directly to local disturbance, while others are due mainly to a general tuberculous toxæmia.

Local.—*Cough* is one of the earliest and most constant symptoms. As a rule, it is at first short, dry, and hacking, but it is soon attended with expectoration. It is often most troublesome at night or in the early morning. In some cases it is paroxysmal and attended with vomiting. *Expectoration* varies much in amount, and is sometimes altogether absent for a time. It is at first mucoid; but it quickly becomes opaque, with streaks or small lumps of purulent matter. As the disease proceeds, the sputa become more abundant; and when cavities have formed, are often compact and nummulated. Microscopic examination reveals tubercle bacilli; * epithelial, pus, and perhaps

* For the method of detecting tubercle bacilli, see Appendix.

some blood cells ; a large amount of granular matter ; and fragments of pulmonary elastic tissue.

Hæmoptysis occurs at some time or other in the majority of cases. It varies from a few streaks to a large quantity of pure blood. In the early stages of the disease, hæmoptysis is rarely severe ; but in later stages, as the result of ulceration of vessels of considerable size, or of rupture of small aneurismal dilatations, it is apt to be very profuse, and may be fatal.

Pleuritic *pains* are of common occurrence. In the earlier stages, they are often referred to the infra-clavicular region ; but later on, they are probably more frequent in the side. Myalgic pains in the chest may also occur from strain in coughing. The breathing is always increased in frequency, but marked dyspnœa (apart from exertion) is unusual.

General.—More or less *general weakness* and loss of energy is always observed. *Fever* is also a constant feature of all cases in actual progress ; and its intensity is in direct proportion to the activity of the disease. Taking, therefore, the course of the disease as a whole, there is great variety of range ; though it often happens that, for long periods, the temperature exhibits remarkable regularity of type, and is essentially *hectic* in nature. When the disease is temporarily quiescent, fever may be altogether absent. In the early stages, also, it may be so slight as to escape detection, unless the temperature be observed at short intervals. A slight rise of evening temperature is often one of the earliest signs, and is of great diagnostic value, especially when associated with a sub-normal morning fall. The *pulse* is rapid and of low pressure ; and is related to the feebleness of the patient rather than to the fever. *Night sweating* is another common toxæmic symptom which is significant of nervous depression, and has little relation to the temperature. *Emaciation* is noticeable

from the first, and its rate of progress affords the best criterion of the general progress of the case. The skin is pale and greasy as a rule. In the later stages *the finger ends* are often *bulbous*, and the *nails* incurved. The *digestive functions* are frequently disturbed, the disturbance being more often of a sub-acid and a tonic type. The appetite is usually bad, with a special dislike for fats; and the bowels are irregular. The *urine* exhibits an absolute excess of solid constituents, altogether out of proportion to the temperature, and in spite also of a diminished appetite for food.

The chief *complications* are pneumothorax from perforation of the pleura, secondary tuberculosis of other parts (especially of the intestines, due to infection from swallowed sputa), fatty degeneration of the liver, fistula in ano, and thrombosis of the femoral veins. Laryngeal tuberculosis is not infrequently associated with pulmonary phthisis.

The *duration and course* of this type of phthisis vary within wide limits. Sometimes, when taken in hand early, the disease may be arrested; sometimes there is great improvement for a longer or shorter time, but the symptoms do not quite disappear; more often, however, the disease makes irregular progress until death occurs from asthenia, or from hæmoptysis, or from some complication.

PHYSICAL SIGNS.—The *thorax* is often small and ill-developed, conforming either to the *alar* or *flattened* type. The earliest signs are usually weakness and slight harshness of inspiration over the apex of one lung, prolongation and more or less bronchial quality of expiration, with perhaps an occasional dry clicking sound or crepitation at the end of inspiration, which may be only heard on coughing. More often at this stage there is no apparent alteration of the percussion-note; or there may be slight dullness from

consolidation, or hyper-resonance from local emphysema. Later on, as consolidation advances, one or other apex may be deficient in movement, or actually flattened by shrinkage of lung tissue; the percussion-note becomes duller, the breath-sounds more obviously bronchial and affecting inspiration as well as expiration, the adventitious sounds more abundant and moister, and the vocal resonance and fremitus are increased. As the area of disease extends, and as softening and excavation develop, we meet with various hollow types of breath-sounds, with loud ringing, crackling, or crepitation, and the vocal resonance becomes bronchophonic or pectoriloquous. The heart is sometimes slightly displaced, and its impulse diffused, owing to shrinking on the part of the lungs; while the heart sounds are heard with unusual distinctness under the clavicles. A systolic bruit is also often heard over the subclavian artery in the same region.

The following are the chief signs of a *cavity*:—A tubular or cracked-pot sound on percussion, especially when the mouth is open; cavernous or amphoric breath-sounds: large hollow metallic or ringing râles, or metallic tinkling; whispering pectoriloquy; and the heart sounds are often intensified, and may acquire a hollow quality by transmission through the cavity.

ACUTE PHTHISIS.

Although pulmonary tuberculosis commonly conforms to the chronic type above described, it is not infrequently acute. However, no rigid distinction can be made between acute and chronic phthisis, as they are so frequently associated. Thus, acute manifestations sometimes usher in the chronic form of the disease, sometimes develop in its course, and sometimes characterise its closing stage. But apart from this, we meet with cases which run a rapid course, and are acute from first to last. Two varieties

may be recognised, which correspond to differences in the mode of infection. In the one it is carried in the blood from some pre-existing tuberculous lesion in the lymph-glands or elsewhere, and gives rise to an *acute miliary tuberculosis* of the lungs. In the other, it is introduced into the air-passages by inhalation, where because of special virulence, or because of special feebleness of resistance, it gives rise to an *acute caseous tuberculosis* of the lung.

ACUTE MILIARY PHTHISIS.—Brief reference has already been made to this condition as a part of general tuberculosis; but a few words may be added here upon the disease as it affects the lungs in particular. It often happens that it is preceded for some time by a period of ill-health. The earliest actual symptoms are cough and expectoration of a bronchitic type. Dyspnœa is also a prominent feature, and may be associated with cyanosis. The temperature is usually high, and is either of a continued type, or exhibits well-marked daily remissions. In some cases the pyrexia reaches its maximum in the morning instead of the evening—a fact of some diagnostic value, when it is present. Hæmoptysis is not common, and it is rarely that tubercle-bacilli can be found in the sputum. The general constitutional disturbance is severe, and often masks the local symptoms. The physical signs are practically indistinguishable from those of acute bronchitis. The disease is uniformly fatal, the duration of the disease varying from 10 or 12 days to a few weeks. The diagnosis is often very difficult. Chief reliance should be placed on the severity of the dyspnœa and constitutional disturbance in comparison with the local signs, on the detection of tuberculosis elsewhere, and on the family and personal history.

ACUTE CASEOUS PHTHISIS.—This form of phthisis, sometimes known as “galloping consumption,” consists of disseminated caseous broncho-pneumonic foci, which

rapidly soften and break down, attended with the destruction of adjacent lung tissue by a suppurative process. Miliary tubercles are seldom to be seen. The bronchi are congested, and stuffed with muco-purulent matter. More or less pleurisy (in some form) is always present. In rare cases, the pneumonic change involves a whole lobe *en masse* (pneumonic type).

The disease may begin abruptly with rigors, followed by high fever; but more often it develops insidiously with cough and expectoration—not infrequently as a sequel of some acute specific fever such as measles, whooping-cough, or influenza. When the disease has developed, the most prominent symptoms are cough, dyspnoea, and profuse expectoration. The latter is for the most part puriform, and sometimes tinged with blood. Serious hæmoptysis is uncommon. The fever runs high, with marked daily remissions. The pulse is frequent, feeble, and of low tension; the tongue is red or furred; the appetite is lost; and the bowels act irregularly. There is great exhaustion, profuse sweating, rapid emaciation, and general restlessness which may pass into actual delirium. The physical signs are those of a rapidly softening broncho-pneumonia. It is probably always fatal; but its duration, though short, is variable.

CHRONIC FIBROID PHTHISIS.

By reason of its slow development and the slight general disturbance to which it gives rise, fibroid phthisis is the most favourable type of pulmonary tuberculosis. Such progress and character are the result of an amount of resistance on the part of the patient, which only *just* falls short of preventing infection altogether. When any predisposition can be traced, it is more often acquired than inherited; and more often local than general. Bronchial

irritation from the habitual inhalation of dust favours the disease. From these facts, it is more common in men than in women ; and in later than in earlier life.

The general nature of the fibroid change has already been referred to. The disease begins at the apex of the lung by the formation of scanty tubercles in the finer bronchi ; which, instead of caseating or setting up broncho-pneumonia, become the starting points of fibrous peri-bronchitis, and general overgrowth of the interstitial tissue, which gradually replaces the normal lung tissue. At the same time, local shrinkage of the fibroid area takes place, with more or less emphysema of the surrounding lung tissue. Small dry caseous masses may be occasionally seen embedded in the fibroid tissue.

The onset is insidious, the course is very slow, and symptoms are relatively few and inconspicuous. There may be some weakness, with loss of flesh, cough, scanty expectoration (if any), and possibly a moderate rise of evening temperature. But all or any of these symptoms are sometimes so slight as to escape notice, unless specially inquired for. The temperature is in the main apyrexial, with a subnormal fall in the early morning ; but from time to time there may be short periods of moderate irregular fever. In other cases the pulmonary symptoms may be more marked as the result of associated bronchitis and emphysema.

The physical signs are often very difficult to distinguish ; but, in typical cases, there is retraction of the chest wall, with limitation of movement, slight dullness on percussion, and weak bronchial breathing over the affected area. Vocal fremitus and resonance are variable. If râles are present, they are due to associated bronchitis. Tubercle bacilli, though not numerous, may usually be discovered by repeated careful examination of the sputa, when the latter are obtainable.

DIAGNOSIS OF PULMONARY TUBERCULOSIS.

The physical signs, taken in conjunction with progressive emaciation, fever, night sweating, cough, mucopurulent expectoration, and occasional hæmoptysis, make the diagnosis of advanced phthisis a fairly easy matter. It is, however, in the earliest stages of the disease that diagnosis is of special importance; and it is here that difficulty is apt to arise. Symptoms, though perhaps individually less characteristic than physical signs, generally precede them; and, in this connection, much value belongs to the quality of *persistence* of even relatively trifling symptoms. Thus undue elevation of the evening temperature, hacking cough on rising in the morning, or steady loss of weight, when persistent and progressive (though slight in degree), should always excite suspicion. The earliest physical signs are weak bronchial or wavy breathing, prolonged expiration, with an occasional clicking sound at the end of deep inspiration after coughing, and slightly impaired (or sometimes increased) resonance at one apex. Again, the detection of tubercle bacilli can now be carried out so readily, that it may be easy, when found, to diagnose cases which would otherwise be obscure. Hæmoptysis, though in itself very suggestive, must not be too hastily regarded as a proof of phthisis. We must be satisfied, first of all, that the blood really comes from the air-passages, and that other causes of hæmoptysis are absent. Hæmoptysis sometimes occurs in nervous young women, or after violent exertion quite apart from phthisis; and the sputa are often streaked with blood after violent coughing from any cause. Alleged hæmoptysis at night only is seldom of pulmonary origin.

But, even in cases where all the above observations have been carefully made, there may still be doubt; and, under these circumstances tuberculin tests may give much help,

though they should never be used as a matter of routine. It may be made by *subcutaneous* injection of tuberculin, when a positive reaction is indicated by general febrile disturbance ; or by superficial inoculation of the skin (von Pirquet) or conjunctiva (Calmette), when reaction is indicated by local inflammation. The first of these methods is the best, and for details of technique refer to the Appendix.

THE PROGNOSIS OF PULMONARY TUBERCULOSIS.

Acute cases of pneumonic phthisis and miliary tubercle are always fatal. In chronic cases the prognosis mainly turns on the rate of progress, early diagnosis, and the extent to which the circumstances of the patient permit of proper and adequate treatment. The slower and quieter the progress, and the earlier the disease is recognised and treated, the better the prospects of recovery or improvement. At the same time, it cannot be ignored, that—from one cause or another—it may be unfortunately impossible for a patient adequately to carry out the treatment he needs, and the prognosis is accordingly worse to that extent. A family history of tuberculosis, previous disease, especially of the lungs or pleura, alcoholic abuse, and repeated hæmoptysis are unfavourable points. The duration of the disease varies so widely in different cases that average estimates are of no value. The marriage of affected persons is unquestionably improper ; and even of those in whom disease appears to have been arrested it is attended with considerable risk, especially in the case of women.

TREATMENT OF PULMONARY TUBERCULOSIS.

Only a brief outline of the main principles and indications for treatment can be given here.

The first object is the *maintenance of nutrition* by abund-

ance of good nutritious food up to the full limit of digestive capacity. The ordinary meals should be supplemented by milk or milk-food, early morning, forenoon, bed-time, and (in some cases) during the night. Alcohol, in the form of malt liquor, or wine, taken with food, may be of service as a means of promoting appetite; but in large quantity it is harmful, especially in the form of spirits. Abundance of *fresh air* is of equal importance; and a plan of treatment conducted almost entirely in open-air has been worked out in recent years with the most encouraging results in suitable cases. Although it is better, if the patient can afford it, to enter an institution specially organised for the purpose, the principles are simple, and can be carried out almost anywhere. Those with active or advanced disease spend practically the whole of their time at rest (lying down) either in the open air, under shelters, or in rooms with widely opened windows; while those in an early stage spend a variable part of each day in active outdoor exercise, and rest in the intervals. The apportionment of rest and exercise turns mainly on two points—the presence or absence of fever, and the strength of the patient. Marked pyrexia and early occurrence of fatigue demand rest, while under contrary conditions exercise is demanded; graded in kind and degree according to opportunity and capacity.

The question of change of *climate* frequently arises. It is too large a subject to be properly considered here in any detail. The leading principles to be kept in view in selecting a locality are: dry soil, pure air, a maximum amount of sunshine, protection from high winds, good drainage, pleasant surroundings; in short, a climate which admits of the patient being as much in the open air as possible. Under no circumstances should advanced cases, very weakly persons, or those suffering from serious complications be sent away from home.

Of late years, the specific treatment of phthisis by subcutaneous injections of tuberculin has made considerable progress, and encouraging results have been obtained. The preparations recommended are New Tuberculin (TR), or Bacillary Emulsion (BE), which is far cheaper and equally satisfactory. The dosage of each is the same. A plan, which is often followed, is to begin with a very small dose, say, $\frac{1}{10,000}$ m.grms. on alternate days, gradually increasing the dose, until slight reaction occurs. Then, after a longer interval, to recommence with a slightly smaller dose, repeating it in slowly increasing amount, twice a week; and, later on, only once a week. It is most suitable for quiet slowly advancing or early cases. The method may be advantageously combined with sanatorium or climatic treatment.

Tonics such as quinine, arsenic, the hypophosphites, or cod-liver oil are often beneficial, especially during the quieter phases of chronic phthisis. Cod-liver oil should always be given in moderate doses to begin with—either in light wine or milk after food. Some tolerate the oil better when given as an emulsion with malt extract. Tonics, and especially cod-liver oil, should not be given if digestion is disturbed. For loss of appetite, fullness or weight after food, a pale but clean tongue, mineral acid with some vegetable bitter, after meals, is often beneficial. For a catarrhal condition of the stomach, with a coated or raw tongue, it is better to give bismuth in combination with bicarbonate of soda and dilute hydrocyanic acid before meals, and an occasional mercurial dose at bed-time. In all such cases the diet requires careful adjustment.

Sweating at night may be often relieved by tepid sponging at bed-time, with water slightly acidulated with vinegar. When very profuse (in later stages) it is due to prostration, and is best dealt with by hot food and alcohol at bed-time, which may be repeated in the night. Medicinally, a pill of

oxide of zinc with extract of belladonna, or a hypodermic injection of atropine, may be given the last thing at night.

The treatment of hæmoptysis has been already described. *Cough* is a symptom which should be treated with discrimination. When it is confined to the early morning, and no more than effective for the expulsion of secretion, it should not be hastily checked by sedatives. When it occurs chiefly on lying down at night, or the first thing in the morning, some warm milk food with alcohol will often promote expectoration, and clear the air-tubes for a considerable time afterwards. For an irritable cough preventing sleep, half a teaspoonful of bicarbonate of soda and a pinch of salt, dissolved in a wineglassful of peppermint water, to be sipped during the night, often succeeds well. A dry irritable cough may also be sometimes relieved by teaspoonful doses of linseed tea, in which some Spanish liquorice has been dissolved. Flying-blisters of small size, or the application of iodine, or the use of stimulating liniments to the chest, are also very useful under similar circumstances. When the secretion is profuse, drugs such as creosote or eucalyptus may be beneficial, either taken internally or in the form of inhalations. Only when the above measures fail should sedatives be resorted to, and then as sparingly as possible.

The primary indication for the treatment of *fever* is absolute rest, with as much cool fresh air as possible. Antipyretics, such as phenazone, acetanilide, etc., do more harm than good; but tepid spongings, supplemented by tonics internally, such as quinine or arsenic, may often be used with advantage.

EMPHYSEMA.

The term *emphysema* has been applied unfortunately to two entirely different conditions: (a) to air in the con-

nective tissue of the lung, or *interlobular emphysema*; (b) to over-distension of the air-vesicles, or *vesicular emphysema*.

INTERLOBULAR EMPHYSEMA is a condition of little clinical importance. Occasionally, as the result of a wound from without, or of a violent *expiratory* effort, one or more air-vesicles are ruptured, and the air escapes into the neighbouring connective tissue, forming on the surface of the lung rows of little bead-like bubbles corresponding with the boundary lines of lobules. They can be removed by pressure. Air thus extravasated may find its way into the mediastinum, or the areolar tissue of the neck; and it is only under these circumstances that interlobular emphysema can be diagnosed during life.

VESICULAR EMPHYSEMA is for the most part a chronic and permanent condition. In this connection it may be laid down with confidence that *permanent* enlargement of the pulmonary air-cells is always associated with degenerative changes, by which their resistance to distension is diminished, and their elasticity is impaired. Under these circumstances, even the normal negative tension operating on the air-cells will suffice to induce emphysema. For instance, with the decay of advancing years, some degree of emphysema is invariable; and, when well marked, is recognised as a definite variety, under the name of senile or atrophous emphysema. Obviously, then, as the result of abnormal distensile strain, the occurrence of emphysema would be rendered still more certain. We find, in this fact, the reason why emphysema is so common an accompaniment of bronchitis and chronic pulmonary disease.

There has been much controversy as to whether local malnutrition is the primary cause of emphysema or not, but the issue is of no practical consequence. What is really important is that local malnutrition is essential to the *permanence* of vesicular over-distension. As will be

pointed out, over-distension occurs sometimes in an acute form, without antecedent structural change, and in such cases, provided the integrity of the air-cells is maintained, it ceases with the cause which gave rise to it.

The causes of over-distension may be *inspiratory*, as in the forced inspiratory distension of the lungs in asthma (whatever may be its precise mechanism). Again, when air is prevented by disease from entering into certain parts of the lung, it is clear that, provided the chest expands to the same extent as in health, increased inspiratory distension will take place in the unaffected parts (vicarious emphysema). But over-distension from increased *expiratory* pressure is of far greater frequency and importance, and this occurs as the result of violent or prolonged cough, and muscular effort. Those parts of the lung which are least supported, such as the anterior borders, and apices, are likely to be most affected under this condition.

VARIETIES.—1. *Acute vesicular emphysema* is a term applied to the over-distension of the lungs which so frequently accompanies asthma, whooping-cough, and violent muscular effort. It is of little clinical significance as a rule, as it subsides with the cause which gave rise to it. When frequently repeated, the circulation is obstructed in the walls of the air-cells; and when, as so often happens in asthma, bronchitis is super-added, local nutrition suffers, and emphysema becomes permanent.

2. *Chronic local emphysema* is characterised by permanent over-distension of local groups of air-cells, and is most commonly associated with fibroid tuberculosis of the lung. The affected area is distended, dry, pale in colour, soft, pits on pressure, and fails to retract on removal of the lung from the body. The vesicles are enlarged, and the inter-alveolar septa are atrophied and may become absorbed, so as to form large bullæ by fusion of adjacent air-cells.

3. *Large-lung emphysema*.—Here the vesicles of the whole,

or a large part of one or both lungs are over-distended, causing an increase both in the bulk of the lungs and in the capacity of the thorax. The lungs are paler, larger, and drier than usual. They have a soft, cushiony feel, and readily pit on pressure. If inflated they recoil to a less extent than usual. The vesicles are obviously enlarged, and not infrequently several are thrown into one, by atrophy and perforation of intervening septa. This variety is generally associated with chronic bronchitis.

4. *Atrophous emphysema*.—This is essentially a disease of advanced life. Both lungs suffer equally, and are smaller, lighter, and drier than normal, and they shrink up on opening the chest. The walls of the air-cells are degenerated, atrophied, and often perforated.

SYMPTOMS.—These are marked in direct proportion to the amount of lung tissue affected. *Dyspnœa* is the most important symptom, and its intensity is in direct proportion to the extent to which the lungs are affected. It is increased by exertion, flatulent distension, and especially by bronchitis, with which it is so commonly associated. Sometimes it is so severe as to cause orthopnœa, and occasionally it is paroxysmal or asthmatic in type. *Cough* is usually present, but is not attended with expectoration apart from co-existent pulmonary or bronchial mischief. The complexion is dusky, general nutrition gradually fails, and the patient is languid and dull. The finger-ends are often clubbed in chronic cases.

Other symptoms of *indirect* origin are met with, which are mainly due to circulatory obstruction in the lungs from destruction, lengthening, and narrowing of the alveolar capillaries. This difficulty is at first counteracted by *hypertrophy* of the right ventricle; but should the heart become over-taxed, symptoms of *cardiac dilatation* will supervene, to be described in another chapter.

The *physical signs* in a marked case of emphysema are

as follows: 1. In the *hypertrophous* form, the chest is enlarged throughout, the ribs are raised, the shoulders high, the sternum thrust forward, and the dorsal spine curved outwards—the chest is “*barrel-shaped*.” In the *atrophous* form the chest is small and long, and the ribs are very oblique. 2. Expansion of the chest is deficient, while elevation movements are in excess. 3. There is hyper-resonance on percussion. 4. The breath-sounds are harsh or exaggerated and divided, and *expiration is prolonged*. 5. Râles (due to co-existing catarrh) are often present, especially over the bases of the lung.

The *prognosis* of chronic emphysema is unfavourable, so far as recovery is concerned, but its progress will depend upon the condition to which it is due.

TREATMENT.—There are two main indications for treatment: first, that of the disease on which emphysema depends, which is generally chronic bronchitis; and secondly, improvement of the general health by good food, tonics, and diffusible stimulants. Among the most useful medicinal remedies are cod-liver oil, tincture of steel, arsenic, and quinine; which may be combined with ether, tincture of lobelia, or spirits of chloroform. Strychnia is a valuable cardiac stimulant in the later stages. Flatulence and constipation demand careful attention. In certain cases, a change to a dry warm climate may be beneficial.

PULMONARY COLLAPSE.

The term “*collapse of lung*” is applied to lung tissue which is simply deprived of air. Collapse is said to be *congenital*, when the lungs have been only imperfectly expanded at the time of birth (*atelectasis*); but it is more often *acquired* in after-life. The latter alone calls for description here. It may arise from *direct pressure on the*

lung, as in copious pleural effusions ; or more commonly as a result of *capillary bronchitis* or *whooping-cough*—a very frequent occurrence with young children.

The mechanism of its production in the latter case, according to Gairdner, is as follows :—The bronchi contain plugs of mucus, which, partly owing to the gradual centrifugal diminution in the calibre of the air tubes, and partly to the greater force of expiration over inspiration, admit the expulsion of air during expiration, but prevent its return during inspiration. But experiments show that wherever a bronchus becomes plugged from any cause, the air imprisoned behind the obstruction is quickly absorbed, and collapse follows ; and the generalisation is probably true, that whenever the air in any portion of lung tissue becomes stationary from any cause—whether from fluid in the pleura, obstructions in the bronchi, or from encroachment on intra-thoracic space by a large heart, by pericardial effusion, or by abdominal distension—absorption of air and collapse will follow. Feeble inspiratory power and the small size of the bronchi sufficiently explain the greater frequency of collapse in children.

Collapse may be limited to a small patch or to the margins of the lung, or involve the entire lung. Collapsed lung tissue is tough and leathery, sinks in water, does not crepitate on pressure, is of a dark slate colour, is depressed on its outer surface below the level of surrounding lung, and when of recent origin may readily be re-inflated. Collapsed lobules are, however, especially apt to undergo inflammatory change, and in that condition can no longer be re-inflated.

The *symptoms* of acquired collapse are not characteristic, and can seldom be dissociated from those of the disease to which it is secondary. With regard to *physical signs*, if the affected area is large, the lower ribs and epigastrium

may be observed to sink inwards during inspiration, and there may be some dullness on percussion, with weak or bronchial breathing. Small areas of collapse cannot be identified during life.

The *treatment* is that for the condition on which the collapse depends. Sometimes in young children, in whom collapse is associated with general bronchitis, great relief is afforded by cold affusions in a hot bath.

GANGRENE OF THE LUNG.

Gangrene of the lung is rare, but it sometimes supervenes in septic forms of broncho-pneumonia ; sometimes as the result of compression of the root of a lung by an aneurism or new growth ; and sometimes from the presence of decomposing particles of food and secretions in the air tubes, especially among the insane and intemperate.

ANATOMICAL CHARACTERS.—Gangrene may be limited to small, well-defined areas, or diffused through a large part of the lung without any line of demarcation. The gangrenous tissue becomes softened, pulpy, of a greenish colour, and horribly foetid. It is usually associated with intense bronchitis, and sometimes with empyema.

SYMPTOMS.—The peculiar and extremely foetid odour of the breath, and the expectoration of dirty brownish or blackish sputa, often containing fragments of lung tissue, are characteristic. Hæmoptysis sometimes occurs. There is great general depression, with low nervous symptoms, and a marked tendency to death from collapse. It is almost always fatal.

There are no *physical signs* peculiar to gangrene, and they are limited to those of bronchitis, consolidation of lung, or empyema with which gangrene may be associated. Sometimes we meet with the signs of a cavity.

It should be remembered that a gangrenous odour of the sputa is sometimes noted in certain cases of chronic empyema, or chronic bronchitis with bronchiectasis, quite apart from gangrene of the lung in mass.

TREATMENT.—Abundant nourishment and stimulants (alcoholic and otherwise) are always necessary. Antiseptic inhalations of creosote, carbolic acid, or turpentine are recommended. Quinine, with mineral acids, may be given internally.

SARCOMA AND CANCER OF THE LUNG.

The commonest new growths in the lungs are cancer and sarcoma; and, though sometimes primary, are much more frequently secondary to growths in the breast, digestive organs, or (in the case of sarcomata) the bones. Primary growths are more common in men, are unilateral, and generally form a large single mass which invades and replaces the normal tissue of the lung. Secondary growths, on the other hand, are more common in women, are apt to affect both lungs, and generally take the form of disseminated nodules. Sometimes, however, the growths are confined to the pleura. New growths of the lung are almost always associated with enlargement of the mediastinal glands, and the cervical glands may be also involved. Pleural effusion is also a common accompaniment, and is often hæmorrhagic.

Corresponding to the irregular distribution of new growths, there is great variety in the symptoms and signs to which tumours of the lung give rise. In the disseminated form there may be no symptoms at all. Or we may meet with cough, dyspnoea, fever, and occasional hæmoptysis, without obvious dullness on percussion, closely resembling acute tuberculosis of lung. In other cases, the signs and symptoms are those of chronic pleural effusion; and, apart

from effusion, a large mass of new growth in the lung may yield physical signs which are very difficult to distinguish from those of a pleural effusion. Lastly, we meet with cases in which the signs and symptoms are those of a mediastinal tumour, with indications of pressure upon adjacent parts. Pain is a very variable symptom ; sometimes it is intense, but it is frequently wanting. In any case the strength gradually fails, and the patient finally dies from exhaustion.

The treatment is purely palliative and symptomatic.

SUB-SECTION IV.

DISEASES OF THE PLEURA.

PLEURISY.

PLEURISY, or pleuritis, signifies an inflammation of the pleural membrane ; and may be *acute* or *chronic* ; *local* or *general*. Although a large majority of cases are undoubtedly of microbic origin, yet our present knowledge does not permit of other than a clinical classification. The various forms of acute or sub-acute pleurisy will be first described, to be followed by some observations on pleurisy of a chronic type.

DRY OR FIBRINOUS PLEURISY.

Pleurisy of this type is for the most part secondary to inflammation of the lung, or thoracic wall ; but cases do occur which appear to be primary and to depend upon chill. Pathologically it is characterised by local congestion, followed by the exudation of lymph ; which, by movement, acquires a rough surface. At this point, after a duration of a few days, it may be reabsorbed, leaving behind a permanent opaque and somewhat thickened spot or patch

to mark the site. In other cases it may lead to permanent fibrous adhesion of the opposed pleural surfaces. And yet again, it may pass on to pleurisy of a sero-fibrinous type.

The symptoms are limited to pain or "stitch" in the side, increased by deep breathing; associated, it may be, with slight cough and pyrexia. A to-and-fro friction sound of respiratory rhythm may often be heard over the seat of pain; and, when present, is very characteristic.

SERO-FIBRINOUS PLEURISY.

These are cases in which dry pleurisy, such as just described, is quickly followed by sero-fibrinous effusion into the pleural cavity. Although not proved in all cases, it is essentially of microbic origin, and more often tuberculous. This is shown by the frequency with which it is followed by phthisis, by the discovery (under special precautions) of tubercle bacilli in the fluid, by the predominance of mono-nuclear cells in the fluid, and by the results of inoculation of guinea-pigs. Certain cases are of pneumococcal or streptococcal origin; in which, however, the effusion is more often purulent. Or the affection may follow on some specific infectious disease. Among predisposing causes, exposure to cold, feeble health, and alcoholism are the most important.

MORBID ANATOMY.—As in "dry" pleurisy, the process begins with congestion and exudation of lymph; but is soon followed by an effusion of sero-fibrinous liquid—clear, or slightly turbid, yellowish, with an average specific gravity of 1,020, and often coagulating to some extent on standing. It has much the same composition as blood serum. The amount varies from a few ounces to four or five pints.

The elastic lung retracts as the effusion increases, and the latter exerts *no direct pressure until the elastic retraction of the lung is exhausted*. In extreme effusions, however,

the lung is *pressed* backwards and inwards against the spine, becoming more or less collapsed if not already inflamed. The opposite lung may be collaterally congested. Similarly, the heart is displaced towards the healthy side—at *first* by the elastic traction of the healthy lung on the mediastinum, and *afterwards* (in large effusions) by direct pressure of the fluid. *The diaphragm, liver, and spleen are never displaced, nor are the intercostal spaces bulged, until the negative pressure on the pleura is exhausted, and replaced by positive pressure.*

The fluid is as a rule gradually reabsorbed, and the fibrinous exudation may also disappear. But, on the other hand, soft connective-tissue granulations may grow into the exudation from beneath, and, when the pleural surfaces come again into contact, ultimately lead to fibrous adhesions. If, while absorption of the fluid proceeds, the re-expansion of the lung is hindered for any reason, the affected side of the chest falls in, and the mediastinum (with the heart) is drawn over to the *same* side. After a variable interval, sometimes extending over many weeks, recovery generally takes place, with or without shrinking of the chest on the affected side.

Symptoms.—The onset of acute pleurisy is frequently insidious, especially in children; but is sometimes sudden and marked by chilliness or actual rigor. The temperature at once begins to rise, but rarely exceeds 103° , and not infrequently falls considerably short of this. Simultaneously, or shortly afterwards, pain, dyspnœa, and cough are observed. This pain is generally referred to the inferior mammary region on the affected side—or, more rarely, to the abdomen, and is sharp, lancinating, and especially aggravated by deep inspiration, or coughing. With the occurrence of effusion, the pain ceases, or is greatly relieved. Dyspnœa is slight as a rule, and is mainly due to the pain in the side and the fever. In fact, when these have sub-

sided, it is remarkable how little the breathing is disturbed, even when the chest is full of fluid, so long as the patient remains quiet. Marked or persistent dyspnoea generally indicates pneumonic complication, or excessive effusion. The cough is short, hacking, and accompanied with no expectoration at first; but later on, as the result of collateral congestion of the opposite lung (especially in large effusions), it may be more troublesome, and accompanied with mucoid expectoration, which is occasionally blood-stained. The decubitus varies much, but more commonly the patient lies on the affected side.

The temperature continues above normal for a variable time, but exhibits no characteristic features; the pulse is frequent, but its ratio to the respiration is only slightly disturbed; the tongue is somewhat furred; the appetite is impaired; thirst is increased; and the bowels are confined.

The course is very variable. An average duration of an ordinary case is from two to three weeks, but it may be much longer, and occasionally it becomes chronic. In exceptional cases the effusion undergoes purulent transformation. Death is a rare result of ordinary pleurisy; but sudden death from syncope sometimes occurs with large effusions, either from exertion, or too rapid withdrawal of fluid during the operation of tapping.

Physical Signs.—Before effusion.—Diminished movement of the affected side, and friction sounds. During effusion:—Enlargement of the side and widening of the intercostal spaces; absent or deficient vocal fremitus; absent or deficient expansion of the side; dullness and increased resistance on percussion over the area of the effusion, beginning at the base and extending upwards, together with a tympanitic percussion-note under the clavicle of the same side; weak or absent breath-sounds, although occasionally they persist, and exhibit a bronchial

quality, especially in children ; absence of friction sounds previously heard ; weak or absent vocal resonance over the fluid, and sometimes ægophony about the upper level of the effusion ; and *signs of cardiac displacement*. In moderate effusions the upper level of dullness is *curved*, with its highest level in or near the axillary region. During absorption the above signs gradually disappear, and the friction sounds return for a while. Should the lung remain unexpanded, the chest-walls on that side retract, the ribs become very oblique, the intercostal spaces very narrow, the shoulder falls, and the angle of the scapula projects. Impaired movement, some dullness and weakness of the breath-sounds usually persist on the affected side for a considerable time.

EMPYEMA.

Empyema, or purulent pleurisy, may be a sequel of the acute sero-fibrinous form, but more often it is purulent from the outset. A purulent effusion indicates greater damage to the pleural membrane than one which is sero-fibrinous, and therefore, as we should expect, empyema is more common in children, whose resistance to damaging influence is feeble. In children, empyema is generally of pneumococcal origin, and is often associated with pneumonia, while in adults it is more often of a streptococcal or septic type, following some infectious fever. Empyema is a most insidious disease, often betraying neither pain, cough, nor dyspnoea, so that it may be easily overlooked, in neglect of physical examination. On the other hand, there is usually some fever of an irregular or hectic type, a peculiar opaque pallor, and general malnutrition. Night sweats are common, and in chronic cases the finger ends are often clubbed. Cough and expectoration are seldom troublesome, apart from complications. Leucocytosis is generally well marked. The physical signs in the main are the same as in the sero-

fibrinous type. A curious sign is sometimes met with, namely, pulsation of the affected side. It appears to be due to a transmitted pulsation of the aorta, in conjunction with a special tension of the effusion. Purulent effusions very rarely undergo natural absorption. If left alone, perforation is apt to occur, either into the lung or externally through the chest-wall. In some cases the pleural cavity may be divided by adhesions into two or more chambers, with imperfect communication between them, each holding pus (*encysted empyema*).

CHRONIC PLEURISY.

Chronic pleurisy, in the form of permanent adhesions and a greatly thickened pleura, is a common sequel of the acute sero-fibrinous variety, and accompanies all chronic inflammations of the lung. Sir A. Clark held that chronic dry pleurisy might be primitive, and become the starting point of progressive interstitial pneumonia, but this view is not always accepted. Pleural effusions may also become chronic, persisting for months without alteration, and such cases are almost always of a tuberculous nature.

DIAGNOSIS OF PLEURISY.

While a careful consideration of the signs and symptoms as above recorded are sufficient, as a rule, to establish the existence of pleurisy, the features which distinguish a pleural effusion from lobar pneumonia demand a brief reference. A pleural effusion always begins at the base of the chest; the upper level of dullness is convex, with its highest point towards the axilla; there is extreme resistance on percussion, and the dullness is absolute; the heart is displaced; and the pulse resp.-ratio is relatively little altered. Lobar pneumonia may involve the apex alone, and when the base is affected, the upper limit of dullness more often

follows the lobar boundary downwards and outwards from the spine ; the dullness and resistance are far less marked ; the heart is not displaced ; and the pulse resp.-ratio is greatly diminished. If there is any doubt, an exploratory puncture should be made with a hypodermic syringe. It must of course be remembered that pneumonia and pleural effusion may co-exist. With regard to the differential diagnosis of individual types of pleurisy, there may be difficulties. It is often impossible to distinguish a purulent from a serous effusion by symptoms and signs alone, though leucocytosis is fairly characteristic. In such cases, exploratory puncture will usually settle the question. Tuberculous effusions may be sero-fibrinous, purulent, or blood-stained, and generally yield negative results on ordinary bacteriological examination. A family history of tuberculosis, prolonged duration of an effusion, persistent return of fluid after tapping, fever (though moderate) of an irregular type with a subnormal early morning fall, night sweats, emaciation, and the detection of tubercle bacilli in the expectoration (if any) are the main points for guidance. Pneumococcal effusions are more often purulent, especially in children, are frequently associated with pneumonia, and pneumococci may generally be found in the fluid. Streptococcal effusions are always purulent, and streptococci are to be found in the fluid. A blood-stained effusion, as distinguished from accidental mixture with blood in the act of paracentesis, points either to tubercle or new growth.

TREATMENT OF PLEURISY.

The *treatment* at the onset should consist of rest in bed, with hot and moist applications to the chest to relieve the pain. If the latter fail, a subcutaneous injection of morphia may be given to an adult, or a few leeches may be applied. Dr. Roberts strongly recommends firm strapping

of the affected side from the onset, with a view of rendering it immobile. The proper action of the skin, bowels, and kidneys should be promoted. To encourage absorption of the effusion—counter-irritation by means of flying-blisters, stupes, sinapisms, or croton oil liniment, together with the administration of iodide of potassium, or pills containing mercury, squills, and digitalis in combination, are recommended; but the action of any mercurial should be closely watched. Under favourable conditions, more often the temperature becomes normal, the fluid is gradually reabsorbed, and convalescence sets in. Bearing in mind that most cases are tuberculous, it is at this point that it becomes so important to restore health and strength by means of fresh air, plenty of good food, reasonable exercise, so that recovery may be thoroughly established before returning to ordinary duties. Alcoholic stimulants are seldom necessary, except in chronic cases where strength is failing.

Paracentesis thoracis, or tapping the chest, may be resorted to for the removal of the fluid when the dyspnœa is urgent, when syncope threatens, when the fluid is purulent, or when the effusion has become chronic. The best method for its performance is by a trocar and cannula fitted with a rubber syphon tube. The best spot to select is the fifth or sixth rib-interspace, in the mid-axillary line. Cough often occurs during the operation, especially when the removal of fluid is too rapid. When it is slight, it will be sufficient to pause awhile, and check the rate of withdrawal of the fluid; but if severe, withdrawal should be stopped altogether. Also if blood begins to appear in the evacuated fluid, or if faintness supervenes, it is well to suspend the withdrawal of fluid. In empyema, free incision of the chest-wall, preferably in the eighth interspace, just outside the angle of the scapula, and with the removal of a small portion of adjacent rib, followed by

thorough drainage, should be invariably practised. Subsequent irrigation is unnecessary, unless the discharge be fœtid. The success of the operation entirely depends upon the re-establishment of contact between the lung and the chest-wall; and consequently, the prognosis is good in proportion to the extent to which the lung can be encouraged to re-expand, and the extent to which the ribs are able to fall in.

HYDROTHORAX—DROPSY OF THE PLEURA.

Hydrothorax only occurs as a part of general dropsy, and hence is generally associated either with Bright's disease or heart disease. It for the most part presents the symptoms and signs of pleurisy with effusion; but it differs from the latter by the absence of all fever, its occurrence on *both* sides of the chest, and the lower specific gravity of the fluid.

The only *symptom* to which it gives rise is dyspnœa, which is marked in proportion to the amount of effusion. The *physical signs* are those of the effusive stage of pleurisy; but there is no friction sound at any time. Its *treatment* is that of the affection on which it depends; but when urgent symptoms are traceable to the effusion, paracentesis may be performed with advantage.

HÆMOTHORAX AND HÆMORRHAGIC PLEURISY.

Hæmorrhage into the pleural cavity, or *hæmothorax*, is usually the result of bursting of an aneurism, or the rupture of a vessel from injury.

Hæmorrhage also occurs in association with inflammation of the pleura, giving rise to a blood-stained sero-fibrinous

effusion (*hæmorrhagic pleurisy*). Such an effusion almost always indicates the presence of some new growth, as tubercle, sarcoma, or cancer. It may be also occasionally met with in connection with low cachectic states, especially in Bright's disease.

PNEUMOTHORAX—AIR IN THE PLEURA.

Air in the pleura is almost always the result of rupture of the pleural surface of the lung, in connection with tuberculous disease. In chronic phthisis, firm pleural adhesions are generally established, which keep pace with the extension of disease. But in acuter developments of a caseous type, before such adhesions have had time to develop, necrotic change close to the visceral pleura may cause the latter to give way, and permit the escape of air into the pleural cavity. Sometimes, however, pneumothorax arises from a wound of the chest-wall, either directly or indirectly, by an associated wound of the lung. The entrance of air is at once followed by retraction of the lung on the affected side, as far as any diseased condition which may happen to be present will permit; and corresponding traction of the opposite lung on the mediastinum will cause displacement of the heart. Pleurisy soon follows, with the effusion of a fluid. The fluid is generally serous at first, gradually becoming sero-purulent, or actually purulent. More rarely it is purulent from the first. When the opening into the pleural cavity is free, the pressure of the contained air corresponds with that of the atmosphere; but when, as the result of a valvular opening, air is drawn in during inspiration and cannot be expelled during expiration, the tension of the contained air may be much raised, giving rise to direct pressure on adjacent parts.

SYMPTOMS.—When the space occupied by air is limited

by pleural adhesions, pneumothorax may be so partial that its occurrence may fail to be recognised. Or, with a very small opening, the development may be gradual. But more often the symptoms usually begin suddenly—possibly during an attack of coughing—with pain in the side, a sense of something having given way, urgent dyspnœa, and general collapse. The pulse becomes frequent, weak, and small; the temperature rises more or less rapidly, though sometimes preceded by an initial fall from shock; and the patient suffers from profound general distress. Usually the more acute symptoms abate in the course of a few hours, but the dyspnœa continues. The subsequent course varies. In traumatic cases recovery may take place. In tuberculous cases, if the patient survive the initial shock and dyspnœa, he usually passes into a condition of chronic pyo-pneumothorax.

The *physical signs*, for reasons already given, are those of both fluid and air in the pleural cavity. The chest is enlarged on the affected side, its movements are diminished, and the heart is displaced to the opposite side; the vocal fremitus and resonance are weak or absent; the percussion-note is tympanitic over the whole area at first; but, as fluid collects, this gives place to dullness at the most dependent part, the upper limit of which is approximately *level*, and *alters with every change of posture* (thus differing from pleuritic effusion); the breath-sounds are weak or absent; metallic tinkling may be observed; and sometimes a splashing sound may be obtained on sharp succussion or shaking of the patient. On placing a coin against the chest-wall (on the affected side), and tapping it with another, a curious ringing sound may sometimes be heard on auscultation, which is very characteristic. With any serious rise of intra-pleural pressure, as the result of a valvular opening, there will be not only increasing displacement of the heart, but marked displacement of the

diaphragm and sub-phrenic viscera, associated with urgent dyspnœa.

DIAGNOSIS.—Pneumothorax can scarcely be confused with advanced emphysema, which always affects both sides of the chest; but a large superficial cavity in the lung or highly marked Skodaic resonance above a pleural effusion may sometimes give rise to difficulty, apart from careful consideration of the history of the case. A limited pneumothorax may easily be altogether overlooked.

The *treatment* in the main is that of pleurisy. A subcutaneous injection of morphia is often of great service at the outset, to diminish the effects of the shock and to relieve the pain. Alcohol often increases the pain. So long as the fluid remains serous or sero-purulent, removal by operation should not be hastily undertaken. Reasonable delay will give a better chance for the opening in the lung to close up. Excessive dyspnœa from intrapleural pressure may be greatly relieved by paracentesis with a small trocar and cannula fitted with a rubber tube, the lower end of which should be passed into a vessel of water. Pyo-pneumothorax must be treated in the same way as empyema.

SECTION VII.

DISEASES OF THE CIRCULATORY SYSTEM.

SUB-SECTION I.

DISEASES OF THE HEART AND PERICARDIUM.

GENERAL CONSIDERATIONS.

WHAT are the factors of efficient circulation? They are partly mechanical, and partly biological. Mechanically, the heart may be regarded as a pump, which propels blood in the direction determined by its valves, into a system of branching elastic tubes, with force sufficient not only to overcome the peripheral resistance opposed to it, but to keep the blood in motion and to bring it back through other tubes to the place from which it started. The questions that arise in this connection relate to the force exerted by the heart, the competency of its valves, the bulk of its output, the progressive increase of the arterial bed at its periphery, and the effect of variations in elasticity and peripheral resistance. Biologically, we have to deal with factors of a nutritional and nervous order. The former are concerned with the maintenance of the structural and functional integrity of the various parts of the circulatory mechanism; and the latter with their control and regulation.

Failure or defect in any one or more of these numerous

factors must inevitably entail circulatory disturbance, proportionate to its extent and duration. This in turn tends to embarrass the heart, to increase its work, and to encroach upon its reserve power.

From these considerations it will be seen that there are three main types of cardiac disease, according as they are primarily or mainly due to mechanical strain, organic disease, or nervous disorder. Although such a classification, broadly interpreted, serves a useful purpose, it must not be pressed too closely, as experience shows many points of close relation between each of the indicated types.

The Heart.—The muscular fibre-cells of the heart differ from those of skeletal muscle, in anastomosing freely with each other, and in the absence of an investing sheath, so that they form a practically continuous structure throughout. Similar continuity is observed in respect of the muscular walls of the two sides of the heart, which are enfolded in such a way that contraction involves corresponding chambers of the heart simultaneously.

The heart-muscle also differs from skeletal muscle in its behaviour. Contraction, for instance, is always maximal, and does not vary with the strength of the stimulus which gives rise to it. For a short time afterwards, no further contraction can be elicited (*refractory period*). An interval of rest is needed for the re-accumulation of contractive power; and consequently, up to a certain point, the force of the next contraction is proportionate to the duration of the pause. Variations in the rate of the heart's action depend on differences in the length of the pause. Accordingly, rapid action implies short pauses, and consequently weakened contraction. Again, in respect of conductivity, the behaviour of cardiac muscle is closely allied with that of nerve-fibres. The property is not evenly diffused over the whole cardiac tissue, but is exhibited in a special degree in a certain

tract, known as the auriculo-ventricular (A-V) bundle. This tract has special structural features, is invested by a delicate sheath, and appears to be a remnant of the primitive cardiac tube (Mackenzie). It rises in the right auricle near the coronary sinus, passes along the A-V septum, and divides into two branches which go to the left and right ventricles respectively; finally breaking up into fine threads (fibres of Purkinje), distributed to the muscular fibre-cells. It is very generally believed, though not conclusively proved, that the impulse of the heart is not primarily of nervous origin (*neurogenic*), but is generated in the cardiac tissue (*myogenic*), starting in the right auricle near the superior vena cava, travelling to the auricles, and thence to the ventricles along the A-V bundle. Accordingly, when the latter is invaded by disease, conduction is apt to be hindered or arrested, and the ventricles then contract irregularly or quite independently of the auricles. Moreover, evidence points to the conclusion that, under certain conditions at present unknown, the cardiac stimulus does not start in the normal place, but elsewhere; and thus gives rise to irregularity in the rhythm of the heart.

Attention may be called also to another property of cardiac muscle which in this case it shares in common with skeletal muscles, namely, *tonicity*. In diastole, the heart is not absolutely relaxed, but is in more or less tonic contraction, sufficient to offer resistance to distension. Diminished tonicity is a factor of great importance in the occurrence of cardiac dilatation.

The influence of the nervous system is concerned with the regulation and control of cardiac function, though it presents many problems which are difficult and obscure. The vagus nerve slows the heart, lowers the blood-pressure, and diminishes conductivity; while, through the sympathetic nerves, the action is quickened, conductivity

improved, and tonicity increased. Both vagi and sympathetic centres are also normally in more or less tonic activity.

Peripheral vessels.—The maximal pressure of the blood in the arteries is reached at the end of the cardiac systole (*systolic pressure*). Owing to peripheral resistance, the larger part is used up in distension of the arteries, but it is restored to the blood-stream by their elastic recoil during diastole; thus converting what would otherwise have been an intermittent into a relatively uniform motion, economising the energy of the heart, and diminishing the strain on peripheral vessels. During diastole, the maximal pressure rapidly falls, and reaches a minimum at its close (*diastolic pressure*). The maximal pressure thus broadly represents the systolic force of the heart; the minimal pressure, the peripheral resistance; and the difference between them, the pulse-pressure, or driving force acting on the blood. The factors of peripheral resistance are twofold—namely, the vaso-motor tone of the arterioles, and the friction exerted by the molecules of the blood on each other and on the walls of the vessels. Each and all of these points require consideration in any estimate of the efficiency of the circulation for a given case.

PHYSICAL EXAMINATION.

Observations under this head must be confined to a few points of special importance. The recognition of the true *apex-beat* of the heart plays such an important part in cardiac topography, that great care should be taken not to confuse it with other movements which may be seen or felt on the chest-wall. Movements transmitted from the right heart are always *retractile*. In dilatation and hypertrophy of the right heart, the true apex is often thrust back from the chest-wall, so that its beat can no longer be felt, and is replaced by retraction movements

which are generally distributed over the sternal ends of the third, fourth, or fifth left interspaces. When the heart is very large, similar retraction may be seen further outwards and downwards. Such movements are also apt to be wrongly interpreted as the result of pericardial adhesion which often gives rise to retractions of a very similar kind.

Cardiac murmurs are audible vibrations of the blood-stream associated with or replacing the normal sounds of the heart. They may arise in different ways—either as the result of organic or structural change in the orifices or valves, causing obstruction to, or permitting regurgitation of, the blood-stream ; or they may be due to deficiency in tonicities of the cardiac muscle, with consequent dilatation, so that a valve is rendered incompetent to close an orifice completely ; or they may occur quite independently of any orificial disturbance. We speak of the first group of murmurs as organic ; of the second, as functional or relative ; and of the third, as accidental.

Murmurs are usually best heard at that part of the chest-wall which is nearest to the seat of production ; thus, as will be seen later on, fixed areas are associated with different murmurs. Then again we are largely guided in diagnosis by the point in the cardiac cycle at which a murmur occurs ; thus we speak of murmurs as systolic, diastolic, presystolic, and so on. Lastly, help may often be derived by noting the direction in which a murmur is best conducted. It is sometimes very difficult to distinguish accidental murmurs from those which are due to orificial or valvular defect. Accidental murmurs most frequently result from anæmia (*hæmic*), or from rhythmical compression of lung tissue adjacent to a forcibly acting heart (*cardio-pulmonary*). Such murmurs are almost always systolic in rhythm, more often heard to the left of the median line, never replacing the first sound, more strictly localised, not associated with obvious

disturbance of the peripheral circulation, and (in the case of card. pulm. murmurs) frequently modified by posture and respiratory movements. Much may often be learnt also by the general condition of the patient.

The arterial pulse.—Very few remarks on this subject are needed for our present purpose. To learn and rightly interpret all that the pulse can teach, requires long practice and experience. Examination by the trained finger will supply all the information that is needed in ordinary practice. A sphygmographic tracing; however, gives the finer details and sequence of events in time which the finger cannot recognise; and provides permanent records. From examination of the pulse we learn the rate, rhythm, and force of the heart; its volume throws light on the cardiac output, when taken in conjunction with the arterial tonus; its compressibility reveals the blood-pressure in general terms; and its resistance (when emptied of blood) suggests the condition of the vessel wall.

The venous pulse.—This can only be properly observed by instrumental means of a rather complicated kind, but a great deal has been lately added to our knowledge of cardiac pathology by its employment. Its special value lies in the fact that it registers all variations of pressure occurring in the right auricle, the behaviour of which is quite inaccessible by other methods of examination. One of the most interesting things it reveals, is the relative frequency with which the auricles are thrown out of action in cases of so-called heart-failure.

For details relating to the pulse—both arterial and venous—the reader is referred to special text-books.

SYMPTOMATOLOGY.

The symptoms which first demand attention are dyspnœa, cough, and hæmoptysis. They are all related to the same cause—namely, stasis of the pulmonary

capillaries dependent upon heart-failure, and especially failure of the left heart. At first, dyspnœa is only complained of during exertion; but even at rest the breathing rate is more often quickened, though the patient may be unconscious of it. As heart-failure increases, dyspnœa recurs on slighter provocation, till it becomes practically continuous. At this stage, orthopnœa is quite common. Paroxysmal attacks of dyspnœa (*cardiac asthma*) may be met with, particularly in cases of aortic incompetence, or cardiac sclerosis. The patient wakes up suddenly in the night, as a rule, with dyspnœa and a sense of suffocation, lasting perhaps for half an hour or more, after which he falls into a restless sleep. In other cases attacks occur by day, generally in connection with effort, as in the act of defæcation. When once established, they are very prone to recur. A third variety of breathing is sometimes observed, namely *Cheyne-Stokes breathing*, especially in elderly men, affected with cardiac sclerosis. It is best seen when the patient is asleep, and consists of short periods of apnœa alternating with laboured breathing. It is often associated with corresponding periodic changes in blood-pressure. It may be so slight, that the patient is unaware of it; or so severe, that he may start up out of sleep gasping for breath, during the apnœic phase. It is closely connected with high blood pressure, but its cause is obscure. It is a sign of grave significance.

Cough and hæmoptysis in cardiac disease call for no special remark beyond pointing out that the latter indicates a high degree of pulmonary stasis; and that, so far from being a cause of anxiety, it often gives great relief from depletion of the engorged vessels.

Sensations varying from simple oppression or discomfort over the precordia to actual pain are also of common occurrence. Their gravity is not in proportion to their severity, but rather to their persistence and the extent

to which they are associated with other signs of circulatory disturbance. The subject of cardiac pain will be considered in more detail later on. It is worth remembering that cardiac patients are apt to show signs of psychic disturbance. More or less mental anxiety is a common feature of cardiac disease. In other cases, bad dreams and some wandering of the mind are observed during the night. More rarely, we meet with actual delusions, or hallucinations of sight and sound.

Other symptoms such as dropsy, serous effusions, and digestive disturbance need only be mentioned here, as they are described elsewhere.

HYPERTROPHY OF THE HEART.

Hypertrophy of the heart is characterised by increased thickness of the heart-wall, which is mainly due to overgrowth of its muscular elements. It cannot be strictly described as a disease in itself, but as a compensatory adaptation rendered necessary by an increase in the work demanded from the heart.

The simplest type of cardiac hypertrophy occurs in healthy persons, from oft-repeated hard muscular exertion, provided it is not carried so far as to exhaust the heart. In such cases we have to deal with a heart of more than average strength. It should be noted that the work which evokes the hypertrophy is intermittent. During the intervals of rest the heart acts quite normally, without any evidence of over-action. In other words, the reserve-power of the heart is increased. If at any time the unwonted exercise is permanently given up, the hypertrophy slowly passes away. In other cases, the increased work demanded from the heart is due to disease, either from an increase in the volume of blood to be propelled, owing to dilatation of the heart cavities;

or from an increase in the peripheral resistance to be overcome, as the result, for instance, of valvular defects, emphysema, or thickening and narrowing of the peripheral arteries. So far as my observation goes, continued nervous excitement of the heart, as in Graves' disease, or from abuse of tea and tobacco, does not directly cause hypertrophy, and when it occurs, it is secondary to antecedent dilatation. In hypertrophy from disease, the cause is persistent, and accordingly the reserve-power of the heart is diminished. Of course it is clear that in any case hypertrophy can only take place when the nutritive conditions of the body are adequate to the occasion. The heart is increased in weight and bulk, not only from increase of its muscular elements, but also from increase in its connective-tissue. Its shape also is modified according to the distribution of the hypertrophy. The latter necessarily varies in its seat and extent according to its cause, but the left ventricle is most frequently affected.

A consideration of the circumstances under which hypertrophy of the heart occurs, shows that it cannot give rise to symptoms. When associated with symptoms of circulatory disturbance, it is because it is insufficient.

The physical signs of hypertrophy of the left ventricle are precordial bulging (rarely), diffusion and displacement of the apex beat downwards and outwards, slow heaving impulse, enlargement of the area of cardiac dullness, prolongation of the first sound, accentuation of the second sound, and a full and tense pulse. The signs of hypertrophy of the right ventricle are not so marked. The apex-beat is displaced to the left, but not downwards. Rhythmical retractions may be observed at the epigastrium, and sometimes in the left rib interspaces; and the cardiac dullness is increased to the right.

The powerful action of the heart in hypertrophy necessarily involves unusual mechanical disturbance and

strain of the valve segments and arterial walls, and may thus lead to morbid changes in these parts.

CARDIAC FAILURE.

A right understanding of what is known as "cardiac failure" is so essential to the proper interpretation of cardiac disease, that it seems desirable, before going further, to devote a chapter to its consideration. A moderate experience of the many forms of circulatory disease at the bedside is sufficient to show that, in spite of their varied clinical history, their closing stages may be reduced to a very few types, characterised by the impairment or arrest of some of the elementary functions of the heart-muscle, on which the efficiency of the circulation primarily depends. This is what is meant by heart-failure.

The same fact may be expressed in another way. Any disturbance of the circulation, however it may be brought about, whether of mechanical, nutritional, or nervous origin, derives its importance from the extent to which it embarrasses the heart, adds to the burden of its work, and encroaches on its reserve-power. When the original cause of disturbance is slight and of temporary duration, complete recovery may be expected. Where, though moderate in degree, it is oft-repeated or persistent, the heart may be able to meet the extra demands made upon it, by means of so-called compensatory hypertrophy of that part of the cardiac wall, on which the stress specially falls; and thus the balance of the circulation may be restored and maintained for an indefinite time. In a third group of cases, where the strain on the heart is so great or so rapidly developed that compensation cannot be effected; or where, either from steady progress of the initial mischief, or the introduction of fresh factors of

disturbance, a previously established compensation is no longer equal to the occasion, the heart becomes exhausted, with impairment or loss of some of its elementary functions. A similar result follows when the heart becomes unequal to the ordinary demands of normal circulation, owing to intrinsic disease of its walls.

As we have seen, the heart has various functions, and it does not necessarily follow that they are all affected at once, or to an equal degree. In fact there is considerable evidence showing that the incidence of failure varies considerably. Theoretically, we may assume at least four different types of heart-failure: (a) failure of contractility, in which the heart either suddenly or by degrees becomes unable to propel its contents onwards; (b) failure of tonicity, characterised by dilatation of heart-cavities, and ultimate engorgement of the systemic veins; (c) failure in conductivity of the cardiac impulse, characterised by slowness or actual stoppage of the heart-beat; and (d) sudden general suspension of cardiac function, under the influence of nervous shock. No doubt in many cases these are variously combined. Unfortunately, our present knowledge is for the most part not sufficiently advanced to permit the classification of individual cases on such an exact basis, although considerable progress is being made in this direction. In actual practice therefore we are obliged to proceed on somewhat broader lines. At the same time, the important fact should be always before us, that the significance of heart disease is not to be measured by evidence of this or that lesion, however prominently its features are forced upon us, but only by the degree of interference with and failure of the elementary functions of the heart.

What is the clinical evidence of cardiac insufficiency? Various tests have been proposed from time to time, based upon alterations of the pulse rate or rhythm, and of the

blood-pressure produced by exertion, and also upon the length of the interval required for the pulse and the blood-pressure to regain their previous condition after disturbance by exertion. But such observations require so many precautions in carrying them out, and are beset by so many fallacies, that they cannot be relied upon, and only serve as useful collateral evidence of failure. As for muscular fatigue in general, the earliest manifestation of cardiac exhaustion is conveyed to the patient himself, by a feeling of distress of some kind. This may take the form of palpitation, shortness of breath, oppression, or other discomfort in the region of the heart or epigastrium, stifling sensations in the throat, or actual pain, which is apt to radiate into the neck and shoulders, and sometimes down the arms. Such symptoms have all the greater significance when they only come on with exertion, and subside on resting. Again, in estimating their importance, careful attention should be paid to the age, general health, occupation, and habits of the patient (not forgetting the disturbing effects of nervousness) in relation to the particular kind and amount of exertion by which the symptoms are elicited. Nor should we too hastily refer such evidence of discomfort directly to any physical abnormality of the heart which may be present, for it may often happen that it largely or even mainly depends upon extrinsic causes, such as overwork, want of food, flatulent distension, loss of sleep, worry, and so on. When these temporary troubles have been dealt with, the heart may be efficient for all practical purposes. Having satisfied ourselves that cardiac failure of some kind exists, the next step should be to ascertain by careful inquiry from the patient and by physical examination the probable way in which it has been brought about, and (as far as possible) the particular cardiac functions which are at fault. Whether, for instance, it is the left or the right heart

which is chiefly in trouble; and whether the failure chiefly involves contractility, tonicity, or conductivity—singly or combined. Although it may not be practicable to carry the analysis of a given case so far as the above remarks would seem to suggest, yet intelligent investigation on these lines is likely to yield useful and important information.

DILATATION OF THE HEART.

Dilatation of one or more of the cavities of the heart is a very common feature of heart-failure. It occurs whenever (during diastole) the intra-cardiac pressure exceeds the resistance of the cardiac wall. It follows therefore that, theoretically, it may be caused either by increased distensile strain, or diminished resistance, or by both combined. Healthy cardiac muscle, however, under the influence of strain, develops increased tonicity, and greater power of resistance, so that pathological dilatation does not and cannot occur until tonicity fails.

The simplest illustration of dilatation is that which is due to exhaustion of tonicity by severe and continued muscular exertion. In its most aggravated form, acute exhaustion of the heart takes place, and fatal dilatation occurs from the greatly increased intra-cardiac pressure. Such an event might happen to one in good previous health; but it would be far more likely in one who was in weak health and out of condition. In cases of milder grade, dilatation (if it occurs) is only temporary; but the heart often remains very sensitive to disturbance from slight strain for a long time afterwards.

In disease, when a chamber of the heart becomes overloaded with blood, as, for instance, in ordinary incompetence of the aortic valve, intra-cardiac pressure is increased, and the resistance of the cardiac wall being

temporarily inadequate, dilatation of the left ventricle takes place sufficient to accommodate the leakage from the aorta. But since the lesion is usually one of slow development, and the heart-wall is relatively healthy, tonicity and contractile power are gradually increased, and a compensatory hypertrophy develops, so that the balance of the circulation is restored, though the dilatation remains. We thus see that dilatation does not *necessarily* point to cardiac weakness. On the other hand, if the lesion continues to progress, or if other disturbing factors arise, hypertrophy becomes inadequate, the heart becomes exhausted, tonicity fails, and pathological dilatation occurs, with symptoms of heart-failure. In acute lesions (as acute myocarditis or rupture of an aortic valve), or when compensatory hypertrophy is impossible owing to myocardial degeneration, progressive dilatation with cardiac failure is liable to set in from the first. At the same time, it is surprising how far myocardial disease may sometimes proceed without any evidence of dilatation. One can only assume that, in such cases, the whole circulation is on a lower grade, so that intra-cardiac pressure is reduced proportionately to diminished cardiac resistance. Nevertheless, there can be no doubt that diminished myocardial resistance must always greatly increase the risk attaching to distensile strain.

Symptoms.—The general effect on the circulation is that the veins become engorged, the arteries imperfectly filled, and the circulation as a whole is retarded. The earliest symptom of dilatation is palpitation, representing a tumultuous struggle on the part of the heart with work to which it is for the time unequal. Dyspnœa occurs about the same time, from interference with the pulmonary circulation; while still later we meet with cyanosis, dropsy, and a variety of other symptoms directly or indirectly due to mechanical congestion of the parts concerned.

The chief *physical signs* are:—diffusion of the apex beat, which has a jerking or slapping quality, and is often irregular or undulating; increased area of cardiac dullness; short, sharp, clear heart-sounds; and a feeble, small, and often irregular pulse. When hypertrophy is combined with dilatation, as so often happens, the signs of each are correspondingly mixed.

The *treatment* of dilatation is practically identical with that required for chronic valvular disease, and its consideration will be deferred to that chapter.

ACUTE MYOCARDITIS.

Many acute febrile diseases are characterised by a special tendency to acute myocardial changes, particularly rheumatic, typhoid, and scarlet fevers, diphtheria, and septicæmia. The lesions are not as a rule evenly distributed, but consist of scattered foci of degeneration affecting the muscular fibre-cells, with localised œdema, and infiltration of leucocytes. The papillary muscles, and the neighbourhood of the auriculo-ventricular ring are particularly prone to suffer. In septic cases small multiple abscesses may be found. The subsequent course depends upon the intensity and duration of the process. In mild cases of short duration, the œdema and exudation may disappear, and recovery take place. If areas of muscle-cells have been actually destroyed, fibroid cicatrices will take their place; or, lastly, a chronic fibrosis and degeneration may result.

The most important effect of acute myocarditis is heart-weakness, with diminished reserve power. So long as the latter is not exceeded, there may be no symptoms, until perhaps some extra strain induces sudden stoppage of the heart. More often there are the usual signs of heart-failure with dilatation, the intensity and persistence of which

will depend upon circumstances. Variations also occur according as the left or right heart is most affected. Or again, sharp attacks of anginal pain may take place on exertion or excitement.

Treatment will be described with that of myocardial degeneration.

MYOCARDIAL DEGENERATION.

Myocardial degeneration is a frequent result of general or local malnutrition. It is more common in the latter part of life ; and it is especially associated with senile decay, severe anæmia, coronary disease, valvular affections, and various forms of toxæmia. Alcoholism and sedentary habits are important predisposing causes.

The chief varieties are three in number: (a) local cicatricial patches; (b) diffuse sclerosis; and (c) fatty degeneration.

Cicatricial patches, composed of fibrous tissue, may be a sequel of acute inflammatory foci, or of small areas of degeneration due to coronary sclerosis. They derive their importance from the fact that they are less resistant than the rest of the heart, and may therefore yield to pressure, forming an aneurismal pouch in the cardiac wall. Also, if a patch happens to involve the A-V bundle, conduction of the cardiac impulse may be hindered or arrested.

Diffuse sclerosis may be met with in senile hearts in association with coronary disease; and also as a result of long-continued dilatation, especially when preceded by hypertrophy. It is characterised by excess of interstitial connective tissue, together with fatty and atrophic changes in many of the muscle-cells.

Fatty degeneration is a common result of severe anæmia, various infective and toxic conditions, myocarditis, and chronic valvular disease. The wall of the left ventricle

is most frequently affected, and its distribution is generally irregular and patchy. The muscular substance is paler than normal, has a greasy feel, and breaks down readily on pressure. The individual fibre-cells lose their striæ, become converted into fat granules, and finally disappear altogether. Fatty degeneration also occurs in connection with fatty overgrowth in and around the heart.

Symptoms of myocardial degeneration.—The symptoms vary a good deal. In one group of cases the countenance is pale and anxious, the temper becomes irritable, the extremities are chilly (especially at night), perspirations break out from slight causes, and sleep is disturbed. The breathing is oppressed and shallow, especially on exertion, and involuntary sighing is often observed. The heart's impulse is weak, and the pulse is feeble, slow (as a rule), and is often persistently irregular both in force and rhythm. From time to time the patient is subject to sudden attacks of faintness, giddiness, or dyspnœa, with or without pain at the chest, in any of which he may die outright. A large proportion of sudden deaths arise from this cause. In a second group sudden death may occur without any suspicion of previous heart trouble; while in a third group the course closely resembles that of chronic valvular disease, with dilatation and dropsy. A diagnosis between fibroid and fatty degeneration is rarely possible. The disease is always serious.

Treatment.—Since in acute myocarditis the cause is often temporary, recovery may be hoped for by a policy of absolute rest, until all symptoms of heart-weakness have subsided; but in chronic degeneration we have to deal with much more persistent mischief, and accordingly treatment is less promising. In all cases, rest is the sheet-anchor of early treatment. When all symptoms of distress have ceased while the patient is at rest, exercise may be permitted—passive to begin with, such as gentle massage

or resistance exercises, gradually leading up to exercise of a more active type. The diet should be light and nutritious; and a small quantity of light wine daily is often helpful. Medicinally, iron and strychnia are useful tonics. Digitalis and drugs of this group may be of great service in certain cases; but they should be given cautiously, and in small doses. Degenerated hearts are peculiarly sensitive to digitalis, and sometimes the drug does more harm than good. Diffusible stimulants and nitrites are indicated in attacks of an asthmatic or anginal nature. Finally the bowels should never be permitted to become confined, but strong purgatives should be avoided.

ENDOCARDITIS.

Endocarditis may be acute or chronic. *Acute* endocarditis probably never occurs apart from infection of some kind, such as rheumatism, scarlet fever, puerperal septicæmia, or gonorrhœa. It is most common in the young. It is convenient to recognise two groups of cases—benign and malignant—although no hard-and-fast line can be drawn between them. Both alike are probably due to the action of micro-organisms, and only differ in the character and degree of inflammatory damage which thus arises. *Chronic* endocarditis is, as a rule, the sequel of the acute form, but it sometimes occurs as the result of persistent mechanical strain.

MORBID ANATOMY.—Endocarditis is more common on the left than on the right side of the heart, and is most marked on the valve segments, to which it is often exclusively confined. In the *acute* form, the endocardium first becomes dull, and sheds its endothelium. Meanwhile the deeper layers of the membrane become infiltrated with leucocytes, and this is followed by small granulations

(*vegetations*) capped with fibrin, which occur for the most part upon the edges or contact-surfaces of the valve-segments or on the chordæ tendineæ. The granulations may increase in size, especially in malignant cases, and form large fungating excrescences, particles of which may be detached and give rise to embolism. Under similar circumstances necrotic changes are liable to occur, followed by ulceration or actual perforation of the valve-segment, or rupture of the chordæ tendineæ. In milder cases the inflammatory changes may subside and pass away, but more often they slowly bring about thickening and puckering of the valve curtains, valvular adhesions, with thickening, shortening, or agglutination of the chordæ tendineæ, or narrowing of the orifices (*chronic endocarditis*).

SYMPTOMS.—*Acute benign endocarditis*, such as so often occurs in the course of acute rheumatism, is apt to come on very insidiously, with few signs or symptoms calling attention to the heart. It is generally first revealed by the detection of a valvular murmur which was not present before. In other cases, however, there may be some evidence of cardiac excitement, palpitation, precordial discomfort, increased frequency of the pulse, dyspnœa, and rise of temperature; but it is very difficult to differentiate such signs and symptoms from those of the primary affection with which it is associated. Mitral murmurs are most common, while aortic murmurs come next in frequency. Recovery is the rule, but the patient is generally left with a permanent valvular murmur.

Acute malignant endocarditis sometimes occurs as the result of a virulent infective element becoming grafted upon an old benign valvular trouble, arising from some grave depression of the general health, or intercurrent toxæmia. In other cases it is malignant from the outset. The symptoms are those of the benign type, *plus* those which are due to infection. The latter admit of twofold

division. On the one hand we meet with symptoms and signs of *general septic infection*, such as irregular fever of a hectic type, rigors, sweating, anæmia, leucocytosis, albuminuria, general prostration, and occasionally delirium, jaundice, and petechiæ. On the other hand, *embolic infarction* is not uncommon, especially in the brain, spleen, kidneys, lungs, or retinae.

Individual cases vary much, but usually conform to one of four types. A *septic* type, in which the condition is one of acute septicæmia, characterised by rigors, sweats, and irregular fever, running a rapid and fatal course, with few cardiac signs or symptoms. A *typhoid* type, so called from the resemblance to the course of typhoid fever, from which its distinction may sometimes be difficult. The type of pyrexia is much the same, the spleen may be enlarged, and diarrhœa is common; but there is leucocytosis, Widal's reaction is negative, and there is no rash. There may be no physical signs beyond those of cardiac weakness and dilatation, but valvular murmurs are more often present, and infarction is of frequent occurrence. The course is fairly rapid, seldom extending beyond a few weeks, when death usually takes place from general toxæmia, heart-failure, or cerebral embolism. A *cerebral* type, in which attention may first be called to the real nature of the case by the occurrence of cerebral embolism, or in which headache, vomiting, delirium, and optic neuritis may supervene as the result of a septic meningitis. Lastly, a *cardiac* type, which is the most common of all, often extending over many months, generally fatal, but sometimes ending in recovery. Such cases are usually secondary to old valvular lesions, which not infrequently have escaped notice, or have been forgotten. The onset is very insidious; and a general feeling of malaise with slight feverishness and increasing weakness may be all that is complained of when the patient first

seeks advice. One or more cardiac murmurs are then discovered, which are apt to change their character from time to time. Embolic troubles are less frequent, but may occur in the later stages of the case. The evidence of toxæmia is limited to hectic fever, night sweats, profound anæmia, and slight albuminuria. Recovery from any form of malignant endocarditis is quite exceptional.

Chronic endocarditis is attended with no general disturbance at all, and is usually overlooked until a valvular murmur is accidentally discovered, or until the patient seeks advice for some failure of compensation. (*Refer to next chapter.*)

TREATMENT.—In all forms of acute endocarditis, prolonged rest in bed is essential—at least from four to six weeks. In acute malignant endocarditis, subcutaneous injection of bacterial vaccines prepared from the patient is worthy of trial; but very often no organism can be detected on examination of the blood. The whole subject is at present in quite an experimental stage. In benign endocarditis of rheumatic origin, it is advantageous (in the writer's opinion) to keep up a steady course of sodium salicylate in combination with sodium bicarbonate and potassium iodide. Cardiac pain or excitement may be soothed by hot applications to the chest-front. In this connection, a small dose of morphia, subcutaneously injected (when not otherwise contra-indicated) is often useful in controlling general restlessness. In threatening dilatation, strychnia in combination with digitalis may be of service. The bowels should be kept open, and the diet during the acute stage should be limited to milk or milk foods. When associated with high tension pulse, an occasional dose of calomel, and drop doses of liq. trinitrin are indicated.

CHRONIC VALVULAR DISEASES OF THE HEART.

Chronic valvular disease may be caused by endocarditis, atheromatous or calcareous degeneration, enlargement of the valvular orifices from excessive cardiac dilatation, or congenital malformation. As the proximate result of valvular disease, one or both of the following defects may occur: *insufficiency*, permitting regurgitation of the blood current owing to a disproportion between the size of the orifice to be closed and the valves to close it, or *obstruction* to the onward passage of the blood from stenosis or partial blocking of the orifice. Each orifice is theoretically liable to both of these defects—*regurgitant* or *obstructive*. As a matter of fact the valves and orifices on the left side of the heart are far more commonly involved than those on the right side, in the proportion of at least three to one. Inflammatory lesions are more frequent in the earlier part of life, as the result of rheumatism, and chiefly affect the mitral orifice and valves; while degenerative lesions are more frequent in the latter half of life, and chiefly affect the aortic orifice and valves. It is of considerable practical importance to note that inflammatory defects often remain stationary for long periods, while degenerative defects are for the most part progressive.

The general progress of chronic valvular disease exhibits a broad uniformity. Four stages may be easily recognised. In the *first stage* the heart is simply embarrassed in its action, thus giving rise to palpitation and dyspnoea on any unusual demand of work, such as bodily exertion. Under favourable conditions, especially in disease of the left heart, this embarrassment is overcome by compensatory hypertrophy (with or without dilatation) of the walls of those chambers of the heart on which strain chiefly falls.

The completeness of compensation depends upon many factors, such as the seat, character, extent, and progress of the valvular lesion, together with the age, state of health, and habits of the patient. When compensation is fairly complete there are no symptoms; but it is often imperfect, and under certain conditions may not occur at all. In no case is it quite permanent, and sooner or later it breaks down, with symptoms of commencing heart-failure and signs of increasing dilatation are observed. The causes of failing compensation vary, but the most common are overwork, malnutrition, nervous exhaustion, intercurrent disease, abuse of alcohol or tobacco, extension of the valvular lesion, pregnancy, over-lactation, and senile decay.

The *second stage* is characterised by functional disorders resulting from mechanical congestion of the parts concerned—disorders of digestion, of absorption, of assimilation, of blood-making, of secretion, and of elimination. Enlargement of the liver and œdema of the ankles and legs are early signs of this stage, because of the low blood-pressure in these parts. Cases in which compensation either does not occur, or is very imperfect, pass directly into this stage at the outset. It often happens that during this period compensation may be re-established by treatment, and all symptoms may disappear for a variable time.

On failure to re-establish or to maintain compensation a *third stage* supervenes, characterised by permanent structural changes in various organs from prolonged congestion, notably in the liver and kidneys, which seriously compromise the general bodily nutrition, and still further weaken the heart. The *fourth and last stage* is manifested clinically by cachexia, from persistent interference with vital functions, and anatomically by degeneration of the body tissues in general, and of the heart in particular.

MITRAL REGURGITATION.—This condition may be pro-

duced by lesions affecting the orifice, valve segments, or cords, as the result of rheumatic endocarditis; or by degenerative changes occurring in later life. It may also occur from defective tonicity and consequent dilatation of the left heart.

Morbid Anatomy.—The valve segments may be thickened, puckered, shrunk, or adherent to each other; vegetations may be present on their faces or edges; and the chordæ tendineæ may be shortened, thickened, deformed, or even ruptured. The left auricle and right ventricle are more or less dilated and hypertrophied, and when regurgitation is considerable the left ventricle is similarly affected. The lungs are congested, and may exhibit hæmorrhages, especially when regurgitation is associated with stenosis. The liver, spleen, and kidneys are enlarged and congested. Thrombi may occur in the heart or veins.

Clinical History.—At each contraction of the left ventricle a certain quantity of blood passes back again into the left auricle, which (together with the pulmonary vessels) thus becomes distended during its diastole in direct proportion to the extent of regurgitation. This necessarily imposes greater work upon both the left auricle and right ventricle during their systole, which (if circumstances are favourable) is followed by hypertrophy of their walls, and thus compensation is established. Hence, during this stage, there are few, if any, manifestations of discomfort; but at the same time, owing to increased fullness of the pulmonary vessels, there is often some dyspnœa on active exertion, and any slight intercurrent catarrh is apt to cause trouble. Further, it must be remembered that, since the auricle is dilated, a larger amount of blood is supplied to the left ventricle at each auricular systole, so that there is also some dilated hypertrophy of the left ventricle. Sooner or later compensation begins to fail from exhaustion of the heart, dilatation of the right

ventricle supervenes, and blood is then forced backwards *through* the pulmonary vessels and right heart to the systemic veins, which now become overloaded; the aorta is underfilled, and the circulation is retarded. The surface of the skin becomes dusky or livid; the ankles are œdematous and the anasarca gradually extends upwards; the liver is enlarged; cough and dyspnœa are well marked, especially on exertion; expectoration is profuse, watery, and sometimes contains blood; and the urine is scanty and is apt to contain albumen. The pulse becomes accelerated, unequal, and usually irregular. The further progress of the case conforms to the general description already given. The *physical signs* are (1) a *murmur* of maximum intensity at the left apex, occurring with or replacing the first sound, conducted outwards into the left axilla, and often heard over the back; (2) accentuation of the second sound at the base of the heart to the left of the sternum; and (3) an occasional systolic thrill over the apex. The murmur is often well heard in the back, between the scapula and the spine.

MITRAL OBSTRUCTION is nearly always due to rheumatic endocarditis, and is often associated with mitral regurgitation. It is essentially a lesion of early life, and is more common in women than in men.

Morbid Anatomy.—The orifice is always narrowed, but its conformation varies. Sometimes, by thickening and shrinkage of the valve-segments, the orifice is reduced to a narrow chink; while in other cases, by adhesion of the valve margins, and thickening of the chordæ tendineæ, it acquires a funnel-shape. From the very beginning of mitral obstruction, increased work is thrown upon the left auricle and right ventricle, which therefore undergo hypertrophy without antecedent dilatation. There is no dilatation until compensation fails owing to cardiac exhaustion. The left ventricle remains unaltered, and

is therefore small in comparison with the hypertrophied right ventricle. Thrombi are often found in the appendix of the left auricle.

Clinical History.—As in mitral regurgitation, there are no symptoms so long as compensation holds good, except slight breathlessness perhaps on unusual exertion; and this immunity may continue for years. With increasing narrowness of the orifice, the heart ultimately shows signs of exhaustion, dilatation of the right heart sets in, accompanied by the usual signs of heart-failure. Hæmoptysis from strain on the pulmonary circulation is not uncommon; and a special liability to embolism should also be noted, due to detached particles of blood-clot from the left auricle.

The *physical signs* vary according to the stage of the disease. During compensation (in uncomplicated cases) the apex-beat is short and sharp, and often displaced somewhat outwards. It may not be felt, owing to backward displacement, from enlargement of the right heart. Undulating retractile movements are often observed over the precordia to the left of the sternum. The area of dullness may be increased. On auscultation a characteristic murmur is heard, *presystolic* in rhythm, running up to and ending in a sharp clear first sound, and often accompanied by a well-marked thrill, which may be felt on palpation. The second sound over the pulmonary artery is accentuated, and often reduplicated. As compensation fails, the second sound is generally inaudible (or nearly so) over the apex; and at a still later stage, the presystolic murmur itself disappears owing to failure of the left auricle. The pulse is small, frequent, and regular as a rule during compensation, but as the heart begins to fail, it is apt to become permanently irregular both in force and rhythm.

AORTIC REGURGITATION occurring in children and

young adults is generally the result of rheumatic inflammation; but in later life it is almost always of degenerative origin, in association with gout, syphilis, alcoholism, or arterio-sclerosis. More rarely it results from rupture of a valve segment.

Morbid Anatomy.—The valve segments are thickened, shrunk, or adherent, and occasionally they are ruptured, perforated, or lacerated. The left heart is greatly enlarged by hypertrophy and dilatation. The coronary arteries are often the seat of degeneration.

Clinical History.—The increased strain brought to bear on the left ventricle during its diastole causes dilatation from the first; but, provided it develops gradually, as is usually the case, the extra propulsive work demanded by the increased contents of the cavity, hypertrophy soon develops; and if the amount of regurgitation be moderate, and the general nutrition of the heart-wall be good, approximate compensation may take place.

Compensation is most stable in cases of stationary regurgitation from endocarditis; and it is surprising for what prolonged periods compensation is often maintained under these circumstances. On the other hand, in cases which depend upon steadily progressive degeneration, the valvular defect increases, until it outruns the capacity for compensating hypertrophy. This is all the more likely to occur when the nutrition of the heart-wall is compromised by degeneration of the coronary arteries.

In some cases, as we have seen, there may be relative or complete absence of symptoms for a long time; while in others, though possibly free from discomfort in repose, palpitation, breathlessness, faintness, and giddiness occur on the least exertion. Cardiac pain, varying greatly in degree, is a common symptom. Disturbance of the cerebral circulation may give rise to throbbing sensations

in the head, subjective disorders of vision or hearing, sleeplessness, and mental disability.

As the result of mechanical strain or progressive dilatation, the mitral valve ultimately becomes involved, and from this point a train of symptoms sets in, similar to what has been already described under the head of mitral regurgitation. Death ultimately occurs from cardiac failure, syncope, apoplexy, or embolism.

The *physical signs* are (1) a more or less heaving and diffused impulse, the apex-beat being always displaced downwards and outwards; (2) increased area of the cardiac dullness; (3) a *murmur* of maximum intensity at the base, following or replacing the second sound, conducted downwards towards the xiphoid cartilage or apex, and also often heard in the back; (4) an occasional diastolic thrill at the base; (5) a *sudden collapsing pulse* with unusual pulsation of the carotids, and in many cases there is capillary pulsation as well. This lesion is very frequently combined with aortic obstruction, and the arteries are often observed to be tortuous and resistant from degenerative changes.

AORTIC OBSTRUCTION.—The etiology of this lesion is similar to that of aortic regurgitation.

Morbid Anatomy.—The valves may become rigid, thickened, and adherent, so that they cannot be pressed back; or the orifice as a whole may be puckered and narrowed. In no form of cardiac valvular disease is pure hypertrophy of the left ventricle better marked.

Clinical History.—In the early stages the symptoms are usually *nil*, for the compensatory hypertrophy of the left ventricle is very complete and persistent, and occurs advantageously *immediately in the rear* of the lesion, for which compensation is needed. After a while the mitral orifice, with its valves, may become implicated, and then the former equilibrium breaks down. Or again,

especially when it happens to be associated with aortic regurgitation, symptoms of discomfort may be present from the outset. The symptoms therefore respectively correspond with those of mitral and aortic regurgitation already described.

The *physical signs* are (1) a heaving diffused impulse which is displaced downwards and outwards; (2) increased area of cardiac dullness; (3) a murmur of maximum intensity at the base, occurring with or replacing the first sound, and conducted upwards towards the large arteries of the neck; (4) an occasional systolic thrill at the base; and (5) a small, steady, but usually regular pulse. An aortic systolic murmur is often heard, especially in old people, which is due merely to roughness or irregularity of the aortic orifice, *without* actual obstruction of the blood-stream. In such cases, of course, there will be no hypertrophy of the left ventricle.

TRICUSPID REGURGITATION.—This occurs either as the result of dilatation of the right heart or of valvulitis. Very moderate distension of the right heart is sufficient to set up a reflux through the tricuspid orifice, so that up to a certain point it may be regarded as a compensatory mechanism to prevent undue engorgement of the heart. In a more marked form, it is a common sequel either of valvular disease on the left side of the heart or of long-standing obstruction in the pulmonary circulation from bronchitis and emphysema. Its morbid anatomy does not differ essentially from that met with in the later stages of mitral disease.

Clinical History.—At each ventricular systole the blood is thrown through the right auricle into the venæ cavæ and jugulars, which speedily become dilated and overloaded. A *reflux pulsation of the jugulars*, which is synchronous with each ventricular contraction, is pathognomonic of tricuspid regurgitation. It therefore follows

that the symptoms which arise from mechanical systemic congestion come on quickly and in an exaggerated form. (See *Mitral regurgitation*.)

The chief *physical sign* of importance is a *systolic murmur*, which is of maximum intensity above the ensiform cartilage. It is not invariably present, and when associated with a mitral systolic murmur, as so frequently happens, their differentiation is very difficult and often impossible.

Tricuspid obstruction and *pulmono-arterial valvular disease* are of such rarity that no special description of them is required in a work of this kind.

DIAGNOSIS OF VALVULAR DISEASE.—The diagnosis of valvular disease is mainly based upon the existence of a *murmur*. An exocardial murmur or friction sound is sometimes liable to be mistaken for an endocardial murmur. Friction sound, however, is mainly distinguished by its “to and fro” rhythm, its superficial grazing or rubbing character, distinct localisation, and absence of any obvious relation to the cardiac sounds. But intra-cardial murmurs are not always due to organic disease of orifices and valves. Hæmic and cardio-pulmonary murmurs may occur, as already described; and dilatation of the left or right ventricle may give rise to mitral or tricuspid regurgitant murmurs respectively, without any structural change in the endocardium. Classification of cases can only be made (1) by a very careful physical examination; (2) by a full inquiry into the previous history, with special reference to rheumatic fever and Bright’s disease, not omitting sex, age, and nature of employment; (3) by a special examination of the superficial arteries and veins for irregularity, intermittence, abnormal pulsations, and indications of atheroma; and (4) by evidence or otherwise of circulatory disturbance, such as irregularity and feebleness of the pulse, cyanosis, or dropsy.

ELEMENTS OF PROGNOSIS IN VALVULAR DISEASE.—The

gravity of a lesion bears no relation either to the intensity or to the quality of a murmur. Functional is less grave than organic disturbance. The nature of the conditions with which a valvular affection is associated or on which it depends, the habits and the employment of the patient, and the presence or absence of pulmonary, renal, or other complications, are of great importance. The condition of the walls of the heart and of the arteries demands the closest attention. Attacks of palpitation, dyspnœa, or precordial discomfort or pain on exertion, a feeble undulating impulse, a liability to syncopal attacks, very scanty and albuminous urine, and anasarca, serve to indicate weakness and degeneration of the former; while inspection and manipulation of the vessels, together with observations as to blood-pressure, give a clue to the condition of the latter. With reference to the relative seriousness of different varieties, it may be said that valvular disease of the right heart is more grave than that of the left; while on the left side the gravity of aortic regurgitation, mitral regurgitation, mitral obstruction and aortic obstruction is in the order enumerated. Sudden death is not a common occurrence, but is most frequent in aortic regurgitation, and is generally due to syncope. Usually death takes place more gradually, as the result of progressive heart-failure.

TREATMENT OF VALVULAR DISEASE.—It should be our object to encourage compensation in every possible way; and on this account everything should be done which tends to improve the general vitality of the patient. When symptoms of cardiac failure supervene, our primary duty is to inquire into and attend to the etiological indication. Thus we must rest a heart that has been overworked, feed a heart that has been starved, soothe a heart that has been worried, enjoin rigid abstinence in those addicted to drink, interdict the use of tobacco in those who are

depressed by resorting to it in excess, adjust the diet in those who have been pampered and over-fed, and so on. But of course while some of the proximate causes of cardiac failure are readily accessible and removable, others are only partially so, and the remainder are beyond control. At the same time the demands upon the physical powers of the heart should be reduced to a minimum, by rest in the recumbent posture in proportion to the gravity of the symptoms; always, however, bearing in mind that so soon as a circulatory equilibrium is realised, and symptoms subside, further rest will only weaken the system, and with it the heart also. It then becomes necessary to encourage the development of cardiac power by carefully graduated exercise. The chief precautions to be observed are these:—(1) Not to attempt it until circulatory equilibrium has been established for the patient when at rest; (2) to be moderate at first; (3) to increase the amount gradually; and (4) always to stop short of fatigue. Slight palpitation and oppression of breathing on beginning treatment need not be seriously regarded.

The food should be light, nutritious, digestible, and not too fluid. No heavy meal should be taken late at night. Alcohol is not needed as a rule in the early stages; but later on, it may be necessary for digestive purposes, for dyspnoëic attacks, extreme feebleness of circulation, or restlessness at night. Tobacco-smoking is generally harmful, and tea and coffee should only be taken in moderation.

Medicinally, cardiac tonics, of which *digitalis* is the chief, are of the greatest value. Dilatation with an irregular and unequal pulse is the leading indication for its use. In favourable cases, the pulse becomes slower, stronger, and more regular, and the urine more abundant. These effects of *digitalis* appear early; and if improvement does not occur within 48 hours, the drug generally fails

altogether. The action of digitalis is enhanced by all measures which relieve cardio-vascular engorgement, and reduce the blood-pressure in the peripheral vessels; and in this connection, the action of calomel deserves special mention. In aortic regurgitation, though digitalis is often of the greatest benefit, yet its administration requires special care, owing to the risk of increasing regurgitation by undue prolongation of the cardiac diastole. It may be stated in general terms, that the healthier the condition of the cardiac substance, the better, *cæteris paribus*, digitalis acts. The best preparations are the tincture and infusion. Full doses are usually desirable in the first instance, to be greatly reduced as soon as the characteristic effects appear. Strychnia, or nux vomica, may often be advantageously combined with digitalis. Strophanthus, caffeine, and convallaria, as heart tonics, are inferior to digitalis, but are useful in alternation with courses of the latter drug, and may be tried when, for any reason, digitalis is not well borne. The peripheral blood-pressure demands close attention, and with a pulse of high tension, mercurial purgatives, iodide of potassium, and the nitrites are often of great service in reducing stress upon the heart.

Dropsy and venous engorgement often demand special depletive measures. For this purpose, hydragogue purgation has a deservedly high reputation, in the form of compound jalap powder ($3\frac{1}{2}$ to 3 j.), or full doses of Epsom salts ($3\frac{1}{2}$ to 3 j. or more) in a limited quantity of fluid. The advantages of dry cupping, leeches over the sternum, or (in urgent cases) venæsection should not be over-looked. Diaphoresis is less desirable, from the risk of undue depression; but, if employed, the cautious hypodermic injection of pilocarpine is preferable to hot air or vapour baths. It is often desirable to puncture the legs for the removal of anasarca.

When once a circulatory equilibrium has been estab-

lished, general massage, good feeding, and tonic remedies are indicated. With evidence of cardiac degeneration, arsenic and iron are of special value.

Acute attacks of palpitation and dyspnœa (cardiac asthma), which not infrequently come on at night, are best combated by large hot fomentations to the chest and administration of small and repeated doses of nitroglycerine, and diffusible stimulants, as spirit and hot water. Sleeplessness is best treated by opiates, unless there is much bronchial secretion, or the renal functions are seriously impaired. Chloral hydrate is dangerous, but bromide of potash, sulphonal, or paraldehyde are unobjectionable. Similar measures are indicated for palpitation and dyspnœa when dependent upon pure cardiac excitement. Threatening syncope demands free recourse to stimulants, together with hypodermic injection of strychnia (℥j. to iv.).

DISTURBANCES OF CARDIAC ACTION.

PALPITATION.—Palpitation may be defined as the conscious sensation of tumultuous action of the heart. It is generally, but not necessarily, associated with increased frequency. It varies from a mere sense of fluttering to the most distressing agitation; but the degree of palpitation affords no measure either of the propulsive power of the heart or of the state of the heart itself. Sometimes it is associated with choking sensations in the throat or a feeling of oppression at the chest. Palpitation on exertion is one of the commonest early indications of a demand of work from the heart in excess of its powers, whether associated with actual disease of the heart or not. Severe over-exertion, especially when accompanied with mental excitement, even in those who have hitherto been in ordinary health, is apt to create a liability to palpitation

from trifling causes (*irritable heart*)—a condition which is very often troublesome to cure. It must not be supposed, however, that palpitation always implies cardiac inadequacy. On the contrary, its commoner and severer forms are more often due to nervous causes, quite independently of heart troubles. It is particularly common in women of nervous habit at the climacteric period of life, and also in hysteria; also from abuse of tea, coffee, or tobacco; or to reflex disturbance from ovarian disease, gastro-intestinal troubles, or constipation.

INCREASED FREQUENCY.—Increased frequency of the heart's action (*tachycardia*), like palpitation, is a common result of cardiac feebleness, whether caused by local mischief or as part and parcel of general debility. Fever is another common cause of undue frequency, roughly to the extent of about ten beats for every degree of pyrexia, but the ratio varies widely. For instance, in scarlet fever it is much exceeded, while in the early stages of typhoid fever it is reduced—facts of some diagnostic value. Persistent undue frequency is also a marked feature of Graves' disease and rheumatoid arthritis. A distinct form of paroxysmal tachycardia is sometimes met with, which differs from ordinary attacks of increased frequency in beginning and ending suddenly, and with a rate which is seldom less than 200 per minute, and often more; whereas in ordinary cases the onset and fall are gradual, and the rate is much slower. The pathology is obscure, but it appears to be often associated with myocardial changes in the region of the auriculo-ventricular ring. The attacks last from a few minutes to many days. In some cases there is no subjective disturbance, while others are attended with considerable distress and oppression in the chest. Although the prognosis is favourable as a rule, long-continued or frequently repeated attacks sometimes lead to serious results.

DIMINISHED FREQUENCY.—Careful auscultation will generally suffice to distinguish genuine slow action of the heart (*bradycardia*) from slowness of the pulse which is due to the failure of immature heart-beats to reach the radial artery. Bradycardia is sometimes habitual, and apparently without pathological significance. In other cases it is due to central or reflex nervous influence acting through the vagus nerve. Such may be the explanation of slow heart-action, for instance, in certain cases of acute infection, cerebral compression, digestive disorders, and pregnancy. In a third group of greater importance, bradycardia is due to myocardial change of some kind, interfering with normal irritability or conductivity; for example, in convalescence from acute fevers, in fatty heart, and in cardio-sclerosis. In this connection, mention should be made of a condition known as *Stokes-Adams disease*, in which a very slow pulse is associated with syncopal or epileptic attacks. It is due to partial or complete arrest in the passage of the impulse from the auricle to the ventricle in the A-V bundle. When partial, only one out of every two, three, or four auricular impulses gets through to the ventricle; and if complete, none at all get through, and the ventricles take up a slow independent rhythm of their own. The nervous seizures are due to cerebral anæmia from imperfect ventricular action.

IRREGULARITY AND INTERMISSION.—Irregularity of the heart's action, like alterations in frequency, may be due to extra- or intra-cardiac causes. Extra-cardiac irregularities are most often the result of undue *nervous* excitability. Thus they are common in children and young persons of neurotic temperament; and, under these circumstances, are apt to exhibit a respiratory rhythm. Another extra-cardiac form of irregularity is connected with irregular filling of the heart, such as may occur as the result of violent respiratory movements,

or pericardial adhesions (*pulsus paradoxus*). Irregularities of this kind are practically confined to alteration in the rhythm of the pulse, and do not affect its force; nor are they attended with any serious disturbance of the circulation.

The simplest form of irregularity from intra-cardiac causes is that which is due to premature heart-beats (*extra-systoles*). The pause which follows each premature beat is prolonged; and since the beat is also immature, the pulse is irregular both in force and rhythm. When the extra-systole is too feeble to raise the aortic valve, there is actual *intermission* of the pulse at the wrist. Extra-systoles may be only occasional, or recur periodically at short intervals. They may be present in pulsation of slow rhythm; and when the action of the heart is quickened by exertion or otherwise, they may temporarily disappear. The condition points to undue *cardiac* excitability; and closer examination shows that there are different types of extra-systole, which probably depend upon variations in the starting-point of the cardiac impulse. Extra-systoles are not uncommon in conditions of high blood-pressure, where the heart is just beginning to fail. On the other hand, they occur and often continue for an indefinite time quite apart from obvious disease, and therefore should not, *per se*, be seriously regarded.

Among other forms of intra-cardiac irregularity, some depend upon defective conductivity, as in partial heart-block; others upon defective contractility, of which the *pulsus alternans* (pulse-waves of larger, alternating with waves of smaller amplitude) is regarded as an example. Lastly, there is a variety which is characterised by persistent and complete arrhythmia, where no two consecutive beats are of the same force or rhythm. This kind of pulse is common in the later stages of mitral disease; and, in many cases at least, appears to be

associated with the condition known as auricular fibrillation. It is characterised by persistent fibrillary twitching of the auricular wall, which no longer contracts as a whole ; and thus the auricular wave is absent from tracings of the venous pulse and electro-cardiograms.

Diagnosis.—It is by no means easy, and not always possible, to distinguish the various forms of cardiac irregularity. Much, however, may be done by careful oral and physical examination, and still more by the employment of modern instruments of precision, directed to the examination of the venous pulse, and electro-motor behaviour of the heart. In general terms, purely nervous derangements only affect the rhythm of the heart ; occur more frequently in young persons of neurotic temperament ; in women than in men ; and in those addicted to abuse of tea, alcohol, and tobacco ; and are not associated with evidence of cardiac disease. On the other hand, irregularity due to intra-cardiac changes are more common in later life ; are more often associated with physical evidence of cardiac lesions ; are more persistent and progressive ; and affect the force as well as the rhythm of the heart.

Treatment.—So long as there are no indications of heart-failure, or of any embarrassment of the general circulation, it is important to encourage and assure the patient in every possible way. He should not be exposed to excitement of any kind, but live a regular and quiet life. A fair amount of active exercise and occupation is most desirable, provided the elements of fatigue and worry are excluded. The diet should be plain and nutritious. Alcohol, tea, and tobacco should be forbidden. Digestive troubles (especially flatulence) should be treated, and the bowels regulated. Probable sources of peripheral irritation should be inquired into, with a view to their correction or removal. Medicinally, tonics are indicated, such as iron, arsenic, and nux vomica. In many cases potassium

bromide is very useful—either given alone, or in combination with one of the preceding drugs.

CARDIAC PAIN—ANGINA PECTORIS.

Pain is far from common in heart disease ; but in certain cases of pericarditis and aortic disease, pain may be a prominent symptom, and appears to be due to direct implication of adjacent nervous structures. But apart from such clearly symptomatic pains, we meet with paroxysmal attacks of pain in and around the heart, and radiating into other parts, which vary widely in their origin, severity, and clinical significance.

As in other functional cardiac disorders, we may recognise organic and nervous types of paroxysmal pains according as they are connected or not with organic disease of the heart substance, but it is by no means always easy to differentiate them.

One of the simplest forms occurs as a purely neuralgic condition in feeble and neurotic subjects, more often in women than in men, and for the most part in early adult life. It is characterised by sharp precordial pain, recurring at short but variable intervals over a considerable period. It is superficial in seat, and it has no relation to exertion, but occurs spontaneously when the body is in repose, and often at night. It is also usually associated with marked tenderness on pressure over the painful area.

Very similar attacks occasionally arise from reflex or peripheral irritation connected with exposure to cold, sudden emotions, flatulent distension of the stomach, or after rapid ingestion of a heavy meal. Such conditions are sometimes referred to as *pseudo-angina*, and are not of serious import.

In another group of cases (*vaso-motor angina*), occupying an intermediate place between neuralgic pseudo-angina

and true angina pectoris, the attacks of cardiac pain seem to be related to a paroxysmal vaso-motor constriction of the peripheral vessels, causing a sudden stress upon the heart in excess of its powers. Probably some other factor is also necessary, such as special nervous irritability. The attacks are often prolonged, and, in addition to pain, are characterised by pallor, cold sweats, chilly extremities, giddiness, tinnitus, and a small pulse. Such attacks are often due to abuse of tobacco, especially when coupled with abuse of alcohol; and may also occur in the course of certain diseases, such as gout, diabetes, chronic malaria, or tabes. For the most part, they cease on removal of the cause; but if cardiac degeneration supervenes, they may gradually merge into true angina.

Angina pectoris is characterised by agonising paroxysms of precordial pain, which are not only accompanied by feelings of distressing anxiety, but are often fatal. It occurs most frequently in men over forty years of age, and is generally associated with disease of the coronary arteries and secondary myocardial degeneration. Its pathology is obscure, but there seems to be no doubt that an anginal attack represents a struggle of a degenerated heart, which is confronted with some temporary strain to which it is unequal.

The *symptoms* of angina present every degree of severity, but in a typical case the pain comes on suddenly, and almost always under the influence of exertion or excitement, is constrictive in character, is usually substernal in site, and often radiates into the arms—especially the left arm down to the elbow. The patient stands still, not daring to move; the face becomes pale and cold, and may be bedewed with sweat. The countenance is expressive of the most intense anxiety, and dread of impending death. The breathing is shallow and frequent, but there is no laboured dyspnœa. The pulse is usually small and thready.

The duration of the attack varies, but it is for the most part quite short. Though a single attack may prove fatal, the earlier attacks are usually followed by recovery, often terminating with some flatulent eructation, with a tendency to recurrence at longer or shorter intervals, until at last a fatal attack occurs. Occasionally the subject of angina suffers from syncopal attacks without pain, in one of which he may die (*angina sine dolore*).

Diagnosis.—True angina may be generally distinguished from the less important neuroses of the heart attended with pain by the following points: It is most common in males between forty and fifty years of age; the attacks are brought on by effort, are rarely nocturnal, are of short duration, and are usually associated with signs of cardiovascular degeneration.

The *treatment* in the milder cases of cardiac pain should be addressed mainly to the removal of the general or toxic conditions on which the pain appears to depend. Temporary relief may be afforded by the use of diffusible stimulants and antispasmodics.

A paroxysm of true angina will generally be best relieved by the use of vaso-dilators, such as nitrite of amyl for inhalation, liq. trinitrin in doses of one or two drops, or brandy and hot water. If, as often happens, these remedies lose their efficiency by repetition, hypodermic injection of morphia may be substituted with advantage. During the intervals between the attacks the patient should be strongly cautioned against hurry or worry of every kind, and the diet should be carefully regulated. The food should be moderate in quantity, with especial restriction of sugars and carbohydrates. The bowels require close attention. It is a good plan to give at intervals—say, once a week—a grain or two of calomel at bedtime, with a saline aperient on the following morning. In gouty cases, iodide and citrate of potash, with some vegetable bitter, thrice daily,

will be found useful; indeed, when the iodide is well borne, it is often very beneficial in other than gouty cases. Arsenic and strychnia are useful tonics, and may be combined with the iodide.

CONGENITAL HEART-DISEASE.

A very brief allusion to congenital heart-disease will be sufficient for our present purpose. For the most part it is characterised by malformations which are clearly the result of arrested development. On the other hand, there are congenital anomalies, especially in connection with the orifice of the pulmonary artery, which are associated with considerable thickening and cicatricial narrowing pointing to inflammatory change. These lesions are attributed by some authorities to ante-natal endocarditis; but, since they are often associated with other defects which are obviously due to arrested development, it is not improbable that the inflammatory changes are secondary to strain and irritation set up by a defect which is primarily developmental.

The more important and frequent congenital anomalies are stenosis of the pulmonary artery at its orifice, defective inter-ventricular septum, patent foramen ovale, and persistent ductus arteriosus.

Though symptoms are sometimes altogether absent, and sometimes delayed in appearance, yet, in the vast majority of cases, we find cyanosis dating from birth or soon afterwards, associated (as the case proceeds) with cough and short breath on exertion, clubbing of fingers and toes, a low temperature, and impaired general nutrition and development. The cyanosis mainly depends upon deficient oxygenation of the blood; but there are doubtless other contributory factors, the exact significance of which

is as yet unknown. Many die early, and few survive to adult life.

ACUTE PERICARDITIS.

Acute pericarditis occurs most frequently as a complication of acute rheumatism, pneumonia, Bright's disease, or some specific fevers; or secondary to new growths such as cancer or tubercle.

The *anatomical changes* resemble, on the whole, those met with in inflammation of the pleura (see Pleurisy). The effusion is sero-fibrinous as a rule; when of pneumococcal or septic origin, it may be purulent; and when associated with new growths, or certain cachectic conditions, it may be bloody. After absorption of the effusion, more or less extensive adhesion is very liable to occur.

SYMPTOMS.—Symptoms may be altogether wanting, and the condition can then only be ascertained by physical examination. In a typical case, the attack is ushered in by pain and oppression over the region of the heart, and palpitation, followed by dyspnoea, fever, and general restlessness. In children, pain and fever are often inconspicuous or absent, and in most cases pain is greatly relieved on the occurrence of effusion. The face is pale and wears an anxious expression, and the decubitus is generally dorsal. The breathing is accelerated and restrained at the outset, becoming more distressed and difficult on the occurrence of effusion, and often attended with orthopnoea. It is accompanied with a dry irritable cough. The pulse is frequent, out of proportion to the breathing; and is liable to sudden variations from slight causes. There is usually more or less nervous disturbance, indicated by headache, sleeplessness, and sometimes delirium or convulsions; and occasionally the nervous symptoms are so prominent as altogether to mask the local affection.

The *physical signs* during the *dry stage* consist of diffused impulse with excited action of the heart. As the result of exudation, we meet in addition with pericardial friction sounds, and occasionally with friction fremitus. In the *stage of effusion* the precordia may be bulged; the apex-beat (if it can be felt) is undulating and irregular in force and rhythm. The area of cardiac dullness is increased, first in an *upward* direction, then laterally. The shape of the dull area is very characteristic, being triangular, the base resting on the diaphragm. Friction sounds may persist throughout the effusion, but they generally become weaker, or disappear altogether. The heart-sounds are feeble, distant, and muffled, and are heard better at the base than at the apex of the heart. In the *stage of absorption*, the cardiac impulse reappears, friction returns, and the area of dullness diminishes in extent.

The distinction of endocardial murmurs from pericardial friction sounds has already been referred to; but it should be remembered that pleural friction may exhibit a cardiac rhythm in the neighbourhood of the heart, which is communicated from the impulse of that organ. The increased area of dullness may be distinguished from that due to cardiac enlargement by the shape of the area, by the presence of dullness at the sternal end of the right fifth interspace (Rotch), and by the absence of such marked impulse as a large heart might be expected to give.

The *course* of the disease varies greatly. It may prove fatal in two or three days with signs of rapid dilatation and heart-failure; or it may run a more protracted course, terminating in recovery with general pericardial adhesion, associated with a large heart and persistent valvular disease; or, again, it may proceed little beyond the dry stage, and quickly recover. In children, as a rule, the onset is insidious, and the symptoms are ill-marked.

The *prognosis* of acute pericarditis without complications

is usually favourable. But it turns largely on the amount of damage which the cardiac muscle has sustained. Or the pericardium may recover, and leave a legacy of chronic cardiac disease behind it. When secondary to acute specific fevers or Bright's disease, it greatly increases the gravity of the associated affection, and often proves fatal.

The *treatment* in the early stages consists of rest, with warm and moist applications to the chest. The pain when severe may be relieved by a hypodermic injection of morphia, or the application of a few leeches to the precordia. Medicines are of little value. The diet should be light and nutritious, and regular action of the bowels should be maintained. When the heart is obviously embarrassed by a large effusion, the latter should be removed by paracentesis. The best and safest point to select for puncture is close up to the sternal end of the fifth left-interspace. During convalescence tonics are called for.

ADHERENT PERICARDIUM.—From a pathological point of view, recovery from acute pericarditis is seldom complete, followed as it is by more or less permanent adhesion of the pericardial surfaces. When the adhesions are partial, and in the absence of endo- or myo-carditic complications, no symptoms or signs occur. But quite often the adhesions are sufficient to embarrass the action of the heart; and, in accordance with principles already described, dilatation and hypertrophy take place, and the heart may attain a large size. It is clear also that these results will be aggravated by disease of the cardiac substance or valves. Compensation is seldom really adequate, and therefore the heart ultimately becomes exhausted, with symptoms of progressive failure. The most important physical signs of adherent pericardium are systolic depression of the intercostal spaces surrounding the apex beat, and sometimes between the eleventh and twelfth ribs behind (Broadbent); immobility of the apex

beat on change of posture; a diastolic rebound of the chest wall in the neighbourhood of the apex; absence of epigastric movement during inspiration from relative fixation of the tendon of the diaphragm, and sometimes a diastolic collapse of the veins at the root of the neck. The area of cardiac dullness is also usually much increased.

SUB-SECTION II.

DISEASES OF THE VESSELS.

ARTERIO-SCLEROSIS.

ARTERIO-SCLEROSIS "is a local or general thickening of the arterial wall with loss of contractility and elasticity, occasioned by fibrous overgrowth mainly of the tunica intima, secondary and proportional to degeneration of the muscular and elastic elements of the media" (Mott). This definition could hardly be improved. The more important predisposing causes are advanced age, over-eating and drinking, persistent over-exertion, infective conditions of various kinds, toxic conditions such as alcoholism and plumbism, and metabolic disorders such as gout and diabetes. As one would expect from these considerations it is more common in men than in women.

Pathology.—Three main varieties are usually recognised—senile, atheromatous, and diffuse. *Senile form.*—Throughout life the arteries undergo progressive thickening. Up to maturity it may be regarded as representing normal vascular growth. Approaching middle age, largely as the result of the general strain of life, connective-tissue overgrowth takes place, with increased thickening of the media and intima. From fifty years of age the media begins to degenerate, with still further thickening of the intima. The latter is probably due to strain caused by

the diminished resistance of the media. Finally calcification is apt to occur, specially affecting the media, and the vessel becomes hard, inelastic, and brittle.

Atheromatous form.—This form is best observed in the aorta, but it is also met with in smaller arteries, especially in those of the brain and heart. It consists of flat, soft elevations of the inner coat, varying in size, rounded in shape, and pearly in colour—afterwards becoming harder and also yellowish from fatty transformation. The endothelial lining usually remains intact. At a later stage the deeper parts of these patches are apt to break up into a soft yellow pulp, forming an atheromatous abscess. More or less calcification is of common occurrence in chronic cases. These changes are associated with hyaline softening of the middle coat, reducing the resistance of the arterial wall, which, but for the proliferative thickening of the inner coat, would be apt to yield and bulge outwards. The local patchy formation points to its dependence upon interference with the blood-supply as the result of morbid changes in the vasa vasorum. The proliferative thickening of the intima is due to strain from local weakness of the arterial wall; and the necrotic changes begin in the central part of the plaques because it is furthest removed from the sources of nutriment.

Diffuse form.—The primary cause of this variety of arterio-sclerosis must be referred to a morbid condition of the blood, which leads to damage of the walls of the smaller arteries and capillaries where structural changes are first observed. These consist, in the first place, of hyaline swelling and degeneration of the capillary walls and middle coat of the arteries. In consequence, the circulation through them is slower and more difficult; the blood-pressure rises; compensatory proliferative thickening of the arterial inner coat takes place; the lumen of the vessel is reduced; and the left ventricle becomes hypertrophied.

At a still later stage degenerative and necrotic changes may take place. The latter, though mainly diffuse in distribution, may be concentrated upon narrowly restricted areas of the arterial wall, and create a liability to rupture and hæmorrhage. Similar changes to those above described affecting the vasa vasorum of the larger arteries account for the frequent association of atheroma with arterio-sclerosis. A general cardio-sclerosis is also apt to be set up when the coronary arteries are involved. Lastly, it is important to note the extremely frequent association of arterio-sclerosis with granular kidney, of which it is a common starting-point.

Symptoms.—It often happens that a long time before there is any evidence of structural change in the vessels, the blood-pressure is persistently high, as the result of vaso-motor constriction dependent on the state of the blood. During this period it is common to meet with irregular and indefinite symptoms, such as dyspeptic troubles, insomnia, giddiness, headaches, mental depression, and general irritability. Even after actual sclerosis has taken place, the symptoms vary greatly according to its intensity and distribution.

The earliest signs of arterio-sclerosis are those which are due to high blood-pressure, namely, accentuation of the aortic second sound; hypertrophy of the left ventricle; an infrequent hard enduring pulse; and a sphygmographic tracing, characterised by a short upstroke, small or absent percussion wave, a broad tidal wave, and a long gradual down-stroke with only a slight dirotic wave.

The symptoms embrace functional derangements from deficient blood-supply, and troubles connected with actual lesions from blocking or actual rupture of vessels. Thus the general nutrition may suffer, as manifested by loss of flesh, a dry pale skin, sensitiveness to cold, a somewhat shrivelled appearance—in point of fact, premature senility.

In other cases, we meet with tingling or itching of the skin, pains in the calves and soles of the feet, a liability to cramp, sudden pains in the muscles on exertion, and sometimes gangrene (*senile gangrene*). When the brain is affected, there may be headache, sleepiness by day and wakefulness by night, rapid fatigue on mental exertion, vertigo, forgetfulness of words, hesitation in speech, epileptiform or apoplectic attacks, cerebral softening from thrombosis, or fleeting paralysis from miliary cerebral hæmorrhage. Dyspnœa of an asthmatic type is not uncommon. When the coronary arteries are involved, there may be anginal attacks, painful palpitation, intermittent or irregular action of the heart and other indications of myocardial change. As circulatory troubles increase, hypertrophy of the left ventricle is replaced by dilatation and symptoms of heart-failure. Finally, chronic renal disease is a very common accompaniment. This brief enumeration of the troubles arising from arterio-sclerosis will suffice to show the clinical importance of the condition.

As to *treatment*, speaking generally, a quiet, equable régime should be enjoined, food and drink should be plain and strictly moderate in quantity, animal food should be largely replaced by cheese, fruit, and vegetables, alcohol (as a rule) should be avoided, and the excretions should be encouraged. Medicinally, a course of iodide of potassium is likely to be useful; while for the relief of many acute functional troubles liquor trinitrin is of great service. The periodic use of hot air or vapour baths is beneficial in many cases. The treatment of secondary affections is described under their respective headings.

SYPHILITIC ARTERITIS.—Syphilis is a frequent cause of arterial disease. It is characterised by localised infiltrations of the deeper layers of the intima with small-celled gummatous tissue, associated with local thickening. In the aorta it forms plaques very like those of atheroma in an

early stage ; but they differ from it by forming tough fibroid patches instead of undergoing caseation and softening. In the smaller arteries, such as those of the brain, it forms small greyish-white nodules or plates, disposed unequally in the circumference of the vessel, and projecting into its lumen, causing more or less obstruction (*obliterative endarteritis*). Accordingly it is very apt to lead to thrombosis. But apart from these direct effects of syphilis, the general devitalising influence of the infection provokes a distinct predisposition to sclerotic changes in later life ; and in this way syphilis may be an active predisposing cause of arterio-sclerosis.

FATTY DEGENERATION OF THE INTIMA is of common occurrence, forming yellow streaks or patches in the aorta and larger arteries. In old people, and in the subjects of pernicious anæmia and phosphorus-poisoning, fatty degeneration of the small arteries and capillaries may occur and lead to hæmorrhage.

AORTIC ANEURISM.

An aneurism is a localised dilatation of an artery, brought about by yielding of some part of the arterial wall. It generally occurs as a result of syphilitic arteritis, or of arterio-sclerosis (atheromatous type) affecting the arterial walls, which accordingly become weakened, and finally give way under the centrifugal pressure of the blood within. Aortic aneurism is much more common in men than in women, and its occurrence is favoured by alcoholism and hard work.

The student is referred to larger textbooks on the subject for a full description of the different varieties of aneurism. Suffice it to say here that the majority of aortic aneurisms are saccular, involving only a part of the circumference of the vessel, and communicating with the artery by an

opening of variable size and shape. The arterial coats, as a rule, can only be traced a short distance into the walls of the aneurism, which are formed for the most part by modification of the structures with which it is in contact. As the result of pressure of the swelling upon surrounding soft parts, they undergo necrosis, the products of which are absorbed as fast as they are formed, and thus the aneurism steadily extends. Hard, resistant structures, such as bone, are gradually also eroded and absorbed. The cavity of the aneurism generally contains more or less fibrin, deposited in layers upon its inner surface, in various degrees of consolidation. The size of the sac varies widely. Its effects mainly depend on pressure and displacement of the structures and organs around it; while as to its results, it generally proves fatal by rupture sooner or later. Sometimes it undergoes a spontaneous cure by coagulation of its contents, and sometimes its progress may be arrested by artificially bringing about a similar result; but such cases are quite exceptional.

SYMPTOMS.—Sometimes no symptoms occur during life, or if present are often so obscure as to render diagnosis very difficult. In such cases, sudden death from rupture of the sac may first reveal the nature of the disease.

THORACIC AORTIC ANEURISM.—The presence of a thoracic aneurism is recognised partly by symptoms, and partly by physical signs, though to an unequal extent in different cases.

The *symptoms* are largely the result of pressure, and therefore are most marked when the aneurism is in close structural relation to surrounding parts. *Pain* is the most constant, and sometimes the only, symptom of aneurism. It varies widely in severity and character, but is always aggravated by exertion. It may depend upon different causes. Sometimes it is due to local pressure or erosion; sometimes to associated coronary sclerosis and

angina with reflected pain in the shoulder and down the inner arm to the elbow, more often on the left side. Very similar arm pain may be caused by direct pressure on nerves of the brachial plexus into the shoulders and down the inner arm to the elbow, more often on the left side.

Pressure on the trachea causes stridor, cough, and dyspnœa. The latter is frequently paroxysmal, closely resembling an attack of asthma. The cough is hard and brassy as a rule, but when the recurrent nerve is involved it is apt to be hoarse, and more or less deficient in tone. Pressure on the root of the lung may give rise to imperfect expansion and weak breath-sounds in that part of the lung which is served by the compressed bronchi, and may ultimately induce collapse or chronic pneumonia. If the pulmonary vein be also compressed, hæmoptysis may occur from congestion; and a similar result follows any leakage of an aneurism into the respiratory passages. Under these circumstances, the condition may be mistaken for phthisis.

Dysphagia and marked emaciation from *pressure upon the œsophagus and thoracic duct* may occur, but are not common.

Pressure on the left recurrent nerve as it winds round the aortic arch is of frequent occurrence, and gives rise to abductor paralysis of the left vocal cord. The right recurrent nerve is seldom involved. *Pressure on the cilio-spinal branches of the sympathetic nerve* accounts for inequality of the pupils.

Pressure on the Blood-vessels.—Compression of the left subclavian or innominate artery may diminish or even obliterate the pulse at one or other wrist. Pressure on the upper vena cava leads to engorgement of its contributory veins, and sometimes to œdema of the neck and face. It is noteworthy that jugular fullness arising in this way differs from that which depends upon dilatation and failure of

the right heart by the fact that it is not reduced by deep inspiration, nor is it associated with venous pulsation. Pressure on the left innominate vein causes distension of the veins over the upper half of the left chest-front.

Physical Signs.—Local bulging and expansile pulsation at some part of the chest-wall may be observed, the seat and degree of which will vary according to the position of the aneurism. On palpation a systolic thrill may be felt over the tumour; and a diastolic shock, when it is present, is of special diagnostic value. As a result of more or less adhesion of an aneurism with the trachea and adjacent mediastinal structures, slight rhythmical tugging of the larynx may often be felt on lightly grasping the cricoid cartilage with a little upward pressure. By percussion, the area of an aneurism may be defined, unless it is too small or too deep-seated. On auscultation, a systolic murmur over an aneurism is quite common, and a double murmur is sometimes heard; but a diastolic murmur alone is quite rare. Special attention should be paid to the seat of maximum intensity, in order to distinguish them from aortic valvular murmurs, which are often associated with thoracic aneurism. A low-pitched ringing accentuation of the aortic second sound is a peculiarly characteristic sign, when present. The heart is not hypertrophied, apart from aortic valvular disease or arterio-sclerosis, but some amount of displacement is not uncommon. A marked difference between the pulse at each wrist is sometimes noted, and may be of much help in diagnosis at an early stage of aneurism. It may be due to one of several causes: pressure on the large vessels as they arise from the aortic arch, the implication of one of them in the aneurismal sac, or partial blocking at their origin by a patch of atheroma or a flake of fibrin. One should not forget, however, that in health the radial pulses may differ, owing to anatomical variations. Sometimes also a lack of synchronism is

observed, between the radial pulses, owing to retardation of the pulse in the arteries arising beyond the aneurism.

With regard to the distinctive features of aneurism according to its position, a few remarks may be made. Aneurisms of the intra-pericardial portion of the aorta are usually small, give rise to few symptoms or signs, and are apt to rupture early. Aneurism of the ascending portion is chiefly manifested by physical signs. Bulging and pulsation, if present, are generally observed at the sternal end of the second right intercostal space. Pressure symptoms are late in appearance, and chiefly affect the vena cava, or root of right lung. The progress is slow, the sac is often of large size, and when rupture occurs it is usually into the right pleural cavity. In aneurism of the transverse portion, pressure-symptoms are prominent and early, but vary according to the part affected; and it is only when the anterior aspect is involved that physical signs are well marked. Rupture is apt to occur early, and the sac rarely reaches a large size. It may rupture into the trachea, left bronchus, left pleural cavity, or externally. Sometimes death occurs from asphyxia. Aneurisms of the descending aorta are so deeply seated that their diagnosis is difficult. Pain in the back may be the only symptom.

The most common affections from which thoracic aneurism must be distinguished are—aortic valvular disease with cardiac hypertrophy and dilatation, mediastinal tumours, and abscesses or new growths in the thoracic wall.

ABDOMINAL ANEURISM may occur in connection with the abdominal aorta or one of its large branches. It may usually be recognised as a pulsating expansile tumour in the abdomen, of rounded shape and very variable size. Like aneurism elsewhere, it causes displacement, compression, or erosion of the parts with which it comes in contact. Among the more common symptoms are pain from compression or tension of nerves; persistent vomit-

ing from pressure on the stomach or bowels ; œdema of the lower half of the body from pressure on the vena cava ; and jaundice from pressure on the bile ducts. The aneurism may ultimately burst into the peritoneal cavity, or one of the hollow viscera.

Undue pulsation of the abdominal aorta is very apt to be confounded with abdominal aneurism. It is more common in early adult life, especially in nervous, hypochondriacal, and dyspeptic subjects. No tumour can be felt, and the pulsation can be traced, on careful examination, along the aorta into the common iliac arteries. It is also associated with nervous palpitation, and an accelerated pulse. Another source of possible confusion may arise from a solid tumour lying over the aorta, and transmitting its pulsations.

The *treatment* of aneurisms (thoracic and abdominal) is very unsatisfactory. Rest should be strictly enjoined ; and only a limited supply of food allowed. To relieve the pain, hot fomentations, lead and opium lotions, or hypodermic injections of morphia may be used. Sedatives, such as chloral, or small doses of opium, are often of value. Medicinally, iodide of potassium, in full doses, is worth a patient trial. The bowels require attention. Various surgical methods have from time to time been practised, such as galvano-puncture, filling the sac with wire, etc. ; but, though individual successes have been attained, the general results are unsatisfactory. Surgical treatment is rarely justifiable.

PHLEBITIS.

ACUTE PHLEBITIS is always associated with thrombosis, but the exact relations in which they stand to each other have been the subject of much difference of opinion. Under the influence of Virchow's teaching it was believed that

phlebitis was usually secondary to thrombosis; but we are now returning to the older view that, in the majority of cases, changes in the vein are antecedent to thrombosis.

Phlebitis may be either *simple* or *suppurative*. *Simple* phlebitis may occur under any one of the following conditions:—the puerperal state, convalescence from acute specific fevers (especially typhoid), grave cachectic conditions associated with anæmic or organic disease, and gout. No doubt in many of these cases there are infective factors in operation, though they may not be obvious. The veins of the lower limbs are most often affected—the left more often than the right. *Suppurative* phlebitis chiefly occurs in connection with traumatic injury of some kind, or with infective trouble in adjacent or closely related parts. Thrombosis of the lateral sinus in mastoid disease, and pylephlebitis from septic foci in some part of the portal tract afford examples of the latter.

SYMPTOMS.—*Simple phlebitis.*—The earliest manifestations are rapidly increasing pain in the limb, together with moderate fever and general malaise. Sometimes the onset is abrupt, and attended with rigor and high fever, but this is unusual. In the case of a large deep vein, swelling of the limb soon follows, beginning at the ankle, and extending upwards. More often, the swelling is soft and pits readily on pressure; but when obstruction involves all the main veins of the limb, as in *phlegmasia alba dolens* after parturition, it is hard and brawny, and does not pit on pressure. The colour of the swollen limb is pale unless the superficial veins are involved, in which case it is dusky. When *only* superficial veins are involved, there is no general swelling, and the affected area often shows diffuse redness. There is marked tenderness in the region of the affected vein, and the latter may often be felt as a resistant cord on palpation. The leg lies quite helpless, and passive movement causes

much pain. The condition remains much the same for two or three weeks, after which gradual improvement usually takes place. Recovery is the rule, but aching and some swelling may persist for an indefinite time. Embolism, from detachment of a fragment of clot, is a possibility always to be kept in mind. Recurrence is not uncommon, especially in cases of gouty origin.

Suppurative phlebitis is more often due to surgical conditions. It need only be stated here that the symptoms are those of pyæmia, coupled with those of the local condition from which it arises.

Treatment.—The main indication is absolute rest in bed, supporting the limb by pillows in the position most comfortable to the patient. The limb should be smeared over with glycerine and belladonna to relieve pain, and then wrapped in cotton wool, secured by an evenly applied bandage. All unnecessary manipulation of the part should be avoided for fear of disturbing the clot, and thus giving rise to embolism. Opiates may be required to relieve pain, and to calm the patient. Food should be simple, but nutritious ; and the bowels should be kept well open. The aching and swelling which persist after acute trouble has subsided will be relieved by bandaging, or an elastic stocking, while the patient is up and about. Under similar circumstances massage is often beneficial.

SUB-SECTION III.

MEDIASTINAL TUMOURS.

NEW growths in the mediastinum are not uncommon. Carcinoma is most frequent, and is more often secondary to similar growth elsewhere. Primary growths are usually sarcomatous ; and, in general frequency, sarcoma ranks next to cancer. Among benign growths, lymphadenoma

is most frequently met with. Mediastinal growths are more common in males than females ; and, as a rule, about middle life. Increasing in size, they implicate the surrounding parts either by extension of the morbid process into their structure, or by exerting pressure upon them, and thus disturbing their functions.

A mediastinal tumour can only be diagnosed with certainty when it has attained some considerable size. The most characteristic features consist of the pressure-signs already described under the head of thoracic aneurism, together with such manifestations of general disturbance as may be peculiar to the growth in question. The diagnosis is further assisted, if we find on physical examination that the mediastinal dullness is increased in breadth, that the heart is displaced, that the glands of the neck are enlarged, or that the original disease is extending to the lungs or pleura, or forming secondary deposits elsewhere. Solid mediastinal tumours differ from aneurism in the absence of expansile pulsation and "blood-murmurs."

The *treatment* is purely palliative.

SECTION VIII.

DISEASES OF THE ALIMENTARY SYSTEM.

SUB-SECTION I.

DISEASES OF THE MOUTH AND THROAT.

STOMATITIS.

Simple stomatitis may be set up by irritation proceeding from over-hot or stimulating food, excessive smoking of tobacco, or certain drugs such as potassium iodide. It is also a frequent accompaniment of dentition, and some specific fevers. The mucous membrane becomes hot and swollen, secretion is increased, and the tongue is coated and often indented at the edge.

Aphthous Stomatitis.—Aphthæ consist of small vesicles, usually few in number, occurring most frequently on the inner aspect of the lower lip or edge of the tongue. The vesicles soon break, leaving small greyish ulcers with bright-red margins, which generally heal in the course of a few days. They may occur at all ages, but are most common in children in connection with acute gastric catarrh. A *recurrent* form is sometimes met with in adults, occurring under obscure conditions, which may be very refractory to treatment. An *infective* form has also been described, which is apparently due to contaminated milk, derived from cows whose udders are similarly affected. It is

attended with considerable fever and constitutional disturbance.

Ulcerative stomatitis is clearly of specific origin, though its precise cause is unknown. It is most common in children whose health has been impaired by bad feeding and dirty surroundings, especially when associated with neglect of the mouth, and the presence of carious teeth. It sometimes occurs in an epidemic form. Ulceration affects the edges of the gums, which become spongy, and bleed on the slightest provocation. It usually begins in and may be confined to the gums of the lower jaw. The ulcers are covered with a dark grey exudate, mixed with blood and pus-cells. Sometimes (though rarely) ulceration extends to the cheek. The lymphatic glands under the lower jaw are very frequently enlarged.

Mercurial stomatitis.—Here, as the name suggests, the inflammation is due to mercurial poisoning, either as the result of a special susceptibility to the drug, or prolonged exposure to its influence from internal administration or otherwise. Adults are more liable to it than children; and it is further favoured by bad teeth, uncleanness of the mouth, and weak general health. Mercury is largely eliminated by the salivary glands, and not only sets up irritation in these glands causing them to swell, but on being carried to the mouth in the saliva gives rise to similar irritation there. Profuse salivation is especially characteristic of this form of stomatitis. The gums become soft, swollen, and spongy, bleeding on the slightest touch; and, in severe cases, ulceration may occur. If administration be promptly stopped, the trouble soon subsides as a rule; but, if unchecked, the teeth may become loose, and (in rare cases) necrosis of the jaw may take place.

Thrush is a form of stomatitis which is associated with the growth of a yeast fungus, named *saccharomyces albicans*. It is a common disorder of children in connection with

digestive derangement, and less frequently it occurs in adults towards the close of long exhausting disease. As a result of fungoid growth, the tongue and mouth become covered with white flaky spots and patches.

Gangrenous stomatitis or *noma* is a rare affection, but is most common in children of weak and delicate habit, living under insanitary conditions, or during convalescence from specific fevers. The first indication of mischief is fœtor of the breath, attended with the formation of a hard swelling in one cheek. The skin outside is red and shining, and on the mucous membrane within is an unhealthy sloughing ulcer. The inflammation includes the whole thickness of the cheek, and then involves the gums and loosens the teeth; and if not checked, the bones are laid bare, the soft parts become gangrenous, and extensive destruction follows. As might be expected, the general disturbance is severe, and marked by fever and profound prostration. It is more often fatal; but, if promptly and thoroughly cauterised, recovery may sometimes take place.

The *symptoms* of stomatitis are much the same in all varieties. Thus, we find pain and soreness of the mouth, which are increased by sucking, mastication, and articulation. In *noma* and some forms of ulceration there may, however, be very little of either pain or soreness. The sense of taste is generally impaired or lost. The breath is almost always unpleasant, and sometimes horribly fœtid. The tongue is usually coated, whether involved in the inflammatory trouble or not. The secretion of saliva is generally increased, and is sometimes profuse. Stomatitis is also very commonly associated with gastro-intestinal disturbance.

TREATMENT OF STOMATITIS.—In all cases, the mouth should be carefully cleansed with antiseptic solutions, of which solutions of borax and chlorate of potash are the most useful. All feeding apparatus should be kept scrupulously

clean. Food should be simple and nutritious ; and the mouth should be washed or wiped out after it. The hygienic surroundings need careful attention. The bowels require regulation. Medicinally, antacids, or rhubarb with soda or magnesia are often of value ; but the drug which is useful above all others, is chlorate of potash—both for local application and for internal administration. In *noma* the slough should be thoroughly eradicated with the electric or thermo-cautery, followed up by employment of an antiseptic mouth-wash, and the internal administration of nutritious food and stimulants.

It is now generally recognised that uncleanness of the mouth, and the multiplication of organisms which is associated with it are a common source of general infection. The condition is one which not only causes by itself impairment of the general health, but one which may seriously affect the progress and behaviour of any infective disease which it happens to accompany, by setting up a mixed infection of a septic type. The accumulation of tartar on the teeth, badly decayed teeth, gingivitis, and pyorrhœa alveolaris are common features. Hence the importance of regular washing of the teeth, cleansing of the mouth, and attention to carious teeth, both as a preventive of disease, and as an essential part of the treatment of all infective disorders, especially those involving the mouth, throat, or nose.

SYMPTOMATIC PAROTITIS.

SYMPTOMATIC PAROTITIS or parotid bubo differs from mumps (p. 94) in the fact that it is not specific or epidemic, and in its proneness to suppurate. It is generally met with as a sequela of typhus, typhoid, or some other specific fever, and is a complication of unfavourable import. It is due to an invasion of the gland by inflammatory micro-

organisms, which reach it by way of Stenson's duct. The neighbourhood of the gland is swollen ; and as suppuration advances, the part may become boggy, and fluctuate on palpation. Pus may be ultimately discharged externally, or into the throat, or external auditory meatus, or it may burrow in the surrounding tissues. The general symptoms are of a low adynamic type.

By way of *treatment* hot fomentations or poultices should be employed, and when the pus has formed it should at once be liberated by incision. A stimulant and supporting plan of treatment is necessary from the outset.

PHARYNGITIS.

ACUTE PHARYNGITIS, or ordinary sore throat, is a catarrhal affection involving all the faucial structures. In some cases it is the result of chill ; while in others it is associated with gouty dyspepsia, or from inhaling some infective matter, as in so-called "hospital sore throat." It usually sets in with chilliness followed by slight fever, which a little later is attended with soreness of the throat and pain in swallowing. Should the inflammation extend into the larynx, there will be cough and huskiness of the voice ; and if into the Eustachian tube, there may be some deafness. The affected mucous membrane is red, dry, and somewhat swollen, or it may be covered with a sticky mucus. The uvula is elongated, the tonsils may be slightly enlarged, and the glands at the angle of the jaw are often swollen and tender. The affection, as a rule, passes off in the course of a few days. The treatment is that already advised for acute nasal catarrh (p. 203).

CHRONIC PHARYNGITIS may be a sequel of the acute form. It is also common in those regularly exposed to weather, especially if associated with alcoholism or over-smoking. The mucous membrane is red and relaxed, and yields a

viscid and tenacious secretion. The lymph follicles are also enlarged and congested, and surrounded by varicose venules. It gives rise to a troublesome hawking cough, which is sometimes accompanied by vomiting. For treatment, nothing is better than electro-cauterisation of the enlarged follicles and varicose vessels.

TONSILITIS.

Inflammation of the tonsils may be acute or chronic. The acute form may be either of a superficial catarrhal, or of a deep-seated suppurative type.

ACUTE TONSILITIS.

Acute tonsilitis is most common in the young, is peculiarly apt to recur, and in some instances it is distinctly epidemic in character. There is also a special liability to tonsilitis in rheumatic subjects. The affection is immediately due to micro-organisms of various kinds, such as staphylo-strepto-, and pneumo-cocci.

1. *Catarrhal tonsilitis* is a superficial inflammation of one or both tonsils. The latter are somewhat enlarged, and the mucous membrane over them is swollen, of a deep red colour, and covered with sticky mucus. The follicles or crypts are filled with catarrhal products, forming yellow spots or patches on the surface. The onset is generally abrupt and characterised by shivering or vomiting, followed by fever and general prostration, in association with pain in the throat and discomfort in swallowing. The voice is stuffy, and articulation is often difficult. The tongue is coated, the breath offensive, and the glands at the angle of the jaw are enlarged and tender. Earache may occur from extension of inflammation up the Eustachian tubes, and albuminuria is not at all uncommon. The constitutional disturbance is considerable, and often out of all

proportion to the local trouble. In the course of about a week the affection usually subsides, but some swelling of the tonsil may persist.

2. In *suppurative tonsillitis* or *quinsy*, the inflammation is more severe, and involves the whole substance of the tonsils. The inflammation may affect one or both tonsils but it is unusual for suppuration to take place in more than one. The tonsil becomes greatly enlarged and congested, and in the course of two or three days, it suppurates, and an abscess is formed. Its follicles become filled with yellowish-coloured plugs consisting of accumulated and altered epithelium. The soft palate and fauces are red, swollen, and more or less displaced by the enlarged tonsil, to which an elongated and œdematous uvula often clings. The disorder is ushered in with chilliness or an actual rigor, quickly followed by high fever (102° — 105°) and the usual signs of febrile disturbance. Simultaneously, or soon afterwards, attention is attracted to the throat, which becomes very sore and painful, especially on attempting to swallow or on separating the jaws. When fluids are taken, they are apt to regurgitate through the nose. If both tonsils are affected, the swelling of the throat may cause difficulty in breathing. The salivary secretion is much increased, and often dribbles from the mouth, the voice is characteristically stuffy, the hearing is impaired, and the glands at the angle of the jaw are always more or less tender and swollen. There is generally considerable depression, the tongue is furred, the appetite is lost, and the bowels are confined. Albumin is often found in the urine, and sometimes in large quantity; but it generally disappears with the other symptoms. In a few days the malady attains its height. The tonsil becomes softened, and fluctuation may usually be detected by the finger, indicating that an abscess has formed. Unless opened artificially it will burst spontaneously somewhat later,

with immediate relief of all symptoms. The prognosis is favourable.

The *treatment* of both forms may be considered together. Milk thickened with some farinaceous material, or simple custard, is the best food, and most easily swallowed. Alcoholic stimulants are often necessary, even from the outset. Ice should be sucked, and hot fomentations may be applied to the neck. Medicinally, much may be done. It is wise to begin with a dose of calomel, followed up by a saline aperient; and small fractional doses of calomel, frequently repeated, are often of much service. For children, a simple saline medicine is indicated, containing ℥ 20-30 of liq. ammon. acet., and ℥ i.-v. (according to age) of tinct. aconite. In rheumatic cases, salicylate of soda is advised. For adults, mist. guaiaci, B.P. (ʒj. every four hours) may be found useful, though it sometimes causes diarrhœa. Gargling the throat with a hot solution of borax is very soothing, when practicable. For great restlessness an opiate may be given at night, such as gr. v.-x. of Dover's powder. In severe cases, it is desirable to anticipate spontaneous rupture of a tonsillar abscess by incision; but it is an operation which requires care. During convalescence tonics may be given, of which tinct. ferri perchlor. is the best.

CHRONIC TONSILITIS.

Chronic enlargement of the tonsils is the essential feature of this condition. It may be a sequel of acute inflammation, or be chronic from the outset. Sometimes it follows on some acute specific fever, especially measles, whooping-cough, and diphtheria; and under these circumstances is very apt to be associated with adenoid overgrowths in the naso-pharynx, and chronic nasal catarrh. On the other hand "adenoids" may occur without obvious en-

largement of the tonsil. Lastly, there are cases in which the tonsillar enlargement cannot be explained.

Chronic enlargement of the tonsils may interfere with health in many ways, especially when combined with adenoid overgrowths in the roof of the pharynx. First of all, it may give rise to local discomfort, and fœtor of the breath from decomposition of tonsillar secretion. It may be attended with obstructive symptoms, such as difficulty in breathing, faulty pronunciation of nasal consonants, and possible chest-deformity from interference with the free entry of air. Then it creates a liability to earache and deafness, from extension of inflammation up the Eustachian tube; also a special liability to repeated "colds." Again, headaches, night-terrors, asthma, and enuresis are examples of reflex nervous disturbance which may be set up by pharyngeal irritation; and lastly, peculiar listlessness and mental dullness are often noted in connection with chronic naso-pharyngeal trouble. If change of air, tonics, and local astringent applications fail to reduce the size of the tonsils, they should unhesitatingly be removed by the knife.

RETRO-PHARYNGEAL ABSCESS.

This somewhat rare affection, as its name implies, is characterised by suppuration behind the pharynx, between it and the vertebral column. It is generally either connected with caries of the spine, or occurs as a sequel of scarlet fever. A soft fluctuating tumour is thus formed at the back of the throat, which modifies the intonation of the voice, and may render swallowing and breathing difficult. If it does not "break" spontaneously, it should be opened by incision.

SUB-SECTION II.

DISEASES OF THE ŒSOPHAGUS.

THE Œsophagus may be *acutely* inflamed by the passage through it of hot, irritating, or corrosive matters, and also in connection with inflammation of adjacent parts (croup, diphtheria, etc.). Pain along the Œsophagus, increased by swallowing, and associated with more or less febrile disturbance, constitute the main features.

The only *chronic* affections that need claim our attention here are those which are attended with obstruction to the passage of food, and thus give rise to *dysphagia*. They may be arranged as follows :

ŒSOPHAGEAL OBSTRUCTION OR STRICTURE MAY DE- PEND ON	{	1. Nervous spasm.	
		2. External pressure	{ Thoracic aneurism. Mediastinal tumour.
		3. Changes in walls	{ Cicatrisation after ulceration. Chronic fibroid thickening.
		4. New growths	{ Cancer. Fibroid polypi.

Spasmodic stricture is characterised by paroxysmal contraction of the muscular coat of the gullet, and is most common in nervous hysterical women. It is sudden in onset, and may be induced by shock, or sudden interruption in the act of swallowing, or as a concomitant of gastric disorder. It affects the swallowing both of solids and liquids. The paroxysm ends as abruptly as it begins, but recurs irregularly on slight provocation, and may be very troublesome to overcome.

The diagnosis of Œsophageal obstruction, due to *outside pressure*, rests upon the conjunction of dysphagia with evidence of thoracic aneurism or mediastinal tumour.

Organic stricture of the Œsophagus usually depends either upon cicatrisation after ulceration caused by corrosive poisons or syphilis (*simple*), or upon cancerous growth

(*malignant*). Simple stricture may occur at any part of the tube ; but malignant stricture commonly affects the upper end, less often the lower end, and the middle very rarely. Upper strictures are generally of the epithelial type, while at the lower end scirrhus is more common. Above the obstruction the tube is dilated, and its walls are thickened ; while below it is collapsed, and its walls are often thinned.

SYMPTOMS.—Dysphagia is the earliest and most striking symptom of œsophageal stricture. It is at first slight, and is only felt on attempting to swallow large solid morsels ; but as the obstruction becomes more complete, both liquids and solids pass with difficulty. Before long, regurgitation of the food takes place, either as soon as it is swallowed, or after an interval, the duration of which depends upon the position of the stricture, and upon the amount of dilatation above it. A good deal of stringy slimy mucus is also regurgitated with the food, together with blood or pus if there be ulceration. Such regurgitation differs from stomach vomiting by its easy spontaneous occurrence, the alkaline reaction of the ejecta, and the absence of any nausea. Pain or discomfort, when present, is generally referred to a point behind the manubrium sterni. Unable to take adequate nourishment, the patient becomes weak and emaciated (though possibly retaining a good appetite), until finally he dies of starvation. The course and progress of any given case obviously depend on the nature of the condition causing the stricture. The average duration of malignant cases is about one year.

DIAGNOSIS.—The use of the bougie will serve to distinguish organic stricture from simple spasm, and obstruction due to external pressure. Simple spasmodic stricture comes on suddenly, runs an irregular intermittent course, and generally occurs in women of an excitable nervous temperament, whereas the course of organic stricture is

steadily progressive. As between simple and malignant stricture, we are guided mainly by the age of the patient, the history of the case, the degree of pain (which is most marked in malignant cases), and the rapidity of progress. Much information may sometimes be gained by careful physical examination of the chest, especially over the posterior mediastinum. The amount of dilatation may be roughly indicated by the interval which elapses before regurgitation occurs.

TREATMENT.—Functional obstruction may sometimes be overcome by simply passing a bougie; but treatment should always be directed to the nervous condition on which it depends. In organic stricture the treatment is very unsatisfactory. Bougies should never be used except for diagnostic purposes (and then only with great caution) where there is any reason to suspect ulceration; but in simple fibroid stricture their systematic use is sometimes beneficial. When the strength fails from want of proper nourishment, nutrient enemata should be given. In some cases gastrostomy has been performed, but without much permanent success.

SUB-SECTION III.

DISEASES OF THE STOMACH.

GENERAL CONSIDERATIONS.

PHYSICAL EXAMINATION.—By physical examination it is possible in the majority of cases to get a very fair idea of the position, size, and shape of the stomach, and also information as to the presence or absence of tumour. In difficult or important cases great help may be afforded by artificial inflation, either through an œsophageal tube or (better still) by the administration of about a teaspoonful of bicarbonate of soda dissolved in water, followed by a

somewhat smaller quantity of tartaric acid also in solution. The latter may be sweetened if desired ; and it is preferable to give only half the quantity of this at first ; and, if the distension is insufficient, the remainder as required. By this means the outline of the stomach is generally indicated with clearness, and the method has the special advantage of enabling a distinction to be readily made between dilatation and downward displacement of the stomach, and also permits the relation of the organ to any tumour in the neighbourhood to be more easily made out. Examination by *x*-rays, after the administration of bismuth carbonate, may also afford valuable information in special cases.

Splashing sounds may be produced in a stomach containing gas and fluid by successive sharp depressions of the abdominal wall over the organ with the points of the fingers. Splashing sounds are not necessarily of morbid significance. They become so if too readily elicited, if unduly persistent, if observed at a time when the stomach should be empty, and if obtained over an abnormal area. Under these circumstances, splashing points to atony of the stomach, with or without dilatation or displacement.

EXAMINATION OF GASTRIC CONTENTS.—In many disorders of the stomach much useful information may be gained by sampling the gastric contents, by means of a flexible rubber stomach-tube. The best time to use the tube is one hour after Ewald's test-breakfast. It should *not* be used in cases of ulcer or advanced cancer of the stomach, of thoracic aneurism, or of severe heart disease, nor in very old people. The test-breakfast consists of two or three ounces of bread with half a pint (or more) of weak tea. The contents of the stomach, removed one hour after such a meal, normally present the following characters. From one to two ounces of a greyish-yellow and somewhat turbid fluid may generally be obtained, which deposits a granular residue of incompletely digested food. It may

be slightly viscid from the presence of a small amount of mucus. On filtration it yields a clear fluid which should be submitted to the following tests :—

(a) *Total Acidity.*—This is made up of free hydrochloric acid, the same acid in combination with proteid substances, organic acids, and acid phosphates. Under normal circumstances the last two are in such small amount that they may be ignored ; but as the result of fermentation organic acids may be present in considerable quantity (lactic, acetic, and butyric acids). The total acidity is estimated by noting the amount of a decinormal solution of caustic soda which is required to neutralise 10 c.c. of the filtered gastric fluid, each c.c. of the soda solution being equivalent to .00365 grm. of HCl. A drop of an alcoholic solution of phenolphthalein having been added to the filtrate, the point of neutralisation is marked by a persistent rose-red coloration. Normally, about 5 c.c. of the soda solution is required, which is equivalent to about .18 per cent. HCl.

(b) *Free HCl.*—Free hydrochloric acid is not found unless there be more acid than is required to saturate and combine with such proteid matter as may be present. If not present, however, one hour after an Ewald test-breakfast, it may be safely assumed that HCl acidity is much reduced. Free HCl is revealed by evaporating to dryness, in a white porcelain dish, a few drops of the filtered contents, with a similar quantity of an alcoholic solution of vanillin and phloroglucin, when (if free HCl be present) a fine red coloration appears at the edges of the residue (Günzburg's test). A still simpler method is to use strips of white filter paper, soaked with Congo-red, and dried. It turns to a sky-blue colour in the presence of free HCl.

(c) *Lactic Acid.*—The presence of this acid in a free state is revealed by the following test : The reagent employed is carbolic acid solution (1 in 10) 10 c.c., distilled water 20

c.c., and liq. ferri perchlor. 1 or 2 drops. The solution has a delicate violet colour, and, on the addition of a small quantity of the gastric filtrate, this colour is replaced by a clear canary-yellow if lactic acid be present (Uffelmann's test).

(d) Observations should also be made with regard to the total quantity of stomach-contents removed, the presence or absence of undigested food, the relative proportion of mucus, and the possible presence of bile or blood.

ESTIMATION OF THE MOTOR FUNCTIONS.—The motor functions of the stomach are concerned partly with continuous churning movements, by which the contents of the stomach are converted, as digestion proceeds, into a more or less uniform chyme, and partly with intermittent expulsion of the contents through the pyloric orifice.

The length of time for which the ingredients of a meal remain in the stomach depends chiefly upon their amount, fluidity, and reaction, and upon the tone and activity of the stomach-wall. The most trustworthy estimates are those which are based upon exploration with the stomach-tube after a meal. The stomach should be empty about two hours after Ewald's test-breakfast, or about five hours after an ordinary dinner meal. If delayed beyond this there is motor insufficiency. "Splashing sounds" afford a useful indication of motor insufficiency when they can be elicited over the whole or greater part of the interval between two consecutive meals.

Various tests of insufficiency have been proposed, consisting of substances which are not acted upon by the acid contents of the stomach, but decompose on their arrival in the bowel, and yield products which are absorbed, and are to be found in the saliva or the urine after an interval of varying duration, according to the time of discharge from the stomach. The results, however, are untrustworthy,

and are attended with practical difficulties, so that they are not of much service.

GASTRIC SYMPTOMS.—1. Pain or discomfort is one of the commonest symptoms of gastric disease. It presents every variety of degree and character, and is generally referred to the region of the stomach itself, behind the sternum, between the shoulders, or to the left side. One of the most important factors in connection with gastric pains is a neurotic or neurasthenic disposition. In persons of this type simple hyperæsthesia and neuralgic affections of the stomach are of common occurrence, and pains arising in other ways become aggravated. Other common causes of pain are distension of the stomach, undue acidity of stomach contents, irritation from indigestible food, and structural disease. Pains arising from visceral displacements, renal or biliary colic, appendicitis, colitis, and intro-abdominal inflammations and growths may be sometimes difficult to distinguish from pains of gastric origin.

The character, seat, and degree of pain cannot be trusted very far for diagnostic purposes. Much more importance attaches to the collateral circumstances under which pains occur, particularly in connection with the taking of food. Brief reference may be made to some of the commoner types.

(a) Pain in the region of the stomach occurring almost immediately after food, irregular in its development, character, and degree, not specially related to any particular food, often quite as bad after fluids as solids, and associated with a nervous temperament, is probably due to hyperæsthesia of the stomach.

(b) Weight and fullness at the epigastrium, beginning soon after food, soon reaching a maximum, and then gradually subsiding, related more to the bulk of a meal than to its ingredients, associated with flatulent eructations, and relieved by the recumbent posture during digestion,

generally depend upon motor insufficiency or atony of the stomach.

(c) Pain beginning one to three hours after food, appearing earlier in light carbohydrate meals than in heavier proteid meals, recurring after meals similar in kind with remarkable punctuality, gradually increasing to a maximum which sometimes culminates in vomiting, or gradually subsiding as digestion comes to an end, relieved by further food or the administration of alkalis, and often associated with radiating pains in the left side or shoulder and neuralgic headache, generally depends on over-acidity. This type is often associated with ulcer of the stomach.

(d) A gradually increasing and progressive pain or discomfort lasting for a few days, aggravated by each meal, and ending with a bulky vomit of highly acid-fermenting material, followed by complete relief, and tending to periodical recurrence, is probably due to pyloric obstruction with dilatation of the stomach.

(e) Pain occurring—so far as can be ascertained—quite independently of food, is more likely to be due to extra-gastric factors than to disorders of the stomach itself.

2. *Flatulence*.—Flatulent distension of the abdomen results from a local or general accumulation of “wind” in the alimentary tract. It is commonly associated with periodic acts of expulsion either by mouth or anus. Flatulent eructation has no constant relation to distension, but is a rather complicated reflex act, closely associated with increased nervous excitability or irritation of the stomach.

The source of “flatus” is not always the same. Sometimes it comes from atmospheric air which is swallowed; and no doubt more or less air enters the stomach with every act of swallowing. Swallowing movements in excess are a common result of throat or stomach irritation in nervous subjects, and many cases of rapid distension of the

stomach occurring independently of food may be thus explained. In other cases, flatus arises from fermentation. The conditions which favour fermentation are undue delay of food in the stomach from atony or pyloric obstruction, diminished HCl acidity, and excess of carbohydrate food. The gas is chiefly carbonic acid, but there is sometimes a variable amount of sulphuretted hydrogen or marsh gas, which gives it an offensive odour.

3. *Water-brash* or *pyrosis* rarely occurs before puberty, and is characterised by a discharge of watery fluid from the mouth, which is often attended with gastric pain and discomfort. It tends to create a feeling of nausea, and often leads on to actual vomiting of the contents of the stomach. The fluid has all the characters of clear saliva, provided it is not accidentally mixed with vomited matter. Water-brash is a paroxysmal salivation dependent upon reflex irritation, sometimes taking its origin in disease of the stomach, sometimes in some irritating article of food as oatmeal, sometimes in disorder of some distant part as the uterus or ovaries. General nervous excitability is also often an important factor in its causation. The amount of fluid which is discharged varies from a mouthful to a half-pint or more.

4. *Heartburn* is the term applied to a disagreeable and peculiar burning sensation along the entire gullet, caused by acrid eructations. It occurs either as the result of excessive acid secretion or of fermentation. In the latter case it is commonly due to butyric acid.

5. *Vomiting* is a reflex act under the control of a nervous centre in the medulla, which may be excited directly by shock, by functional or organic nervous disease, or by toxic conditions of the blood. Much more frequently, however, vomiting depends upon peripheral irritation of some kind, chiefly in connection with disease or disorder of the abdominal viscera, and particularly with the stomach. Gastric

vomiting for the most part depends upon errors in the quantity or quality of food, hydrochloric over-acidity, pyloric obstruction, gastric hyperæsthesia, or structural changes in the walls of the stomach.

When vomiting is slow in development or difficult, it is apt to be preceded by a *feeling of sickness* or *nausea*, associated with pallor, clammy sweats, giddiness, and general depression. Nausea is a feature of vomiting due to peripheral causes in a majority of cases, and in some forms of nervous vomiting, but it is more often absent when associated with organic intra-cranial lesions.

Infants and young children vomit much more readily than adults—a fact which is partly related to their greater reflex excitability, and partly to the more vertical position of the stomach. Taken by itself, therefore, vomiting has not quite the same morbid significance as in adults.

In investigating the cause of vomiting, inquiry should first be made as to the time and circumstance of its occurrence. If rejection occurs immediately, or in a very short time after food, the possibility of regurgitation due to œsophageal obstruction should be remembered. In this connection the reaction affords help in diagnosis. If the rejected matter comes from the stomach it will be acid, but not if it comes from the œsophagus. Otherwise, vomiting which only occurs when food is taken, is due either to morbid irritability of the stomach, or to the food itself. If it occurs at periodic intervals of considerable duration, and is abundant, frothy, and fermentative, pyloric obstruction is almost always indicated. On the other hand, when vomiting occurs repeatedly at irregular intervals, and quite independently of food, it is suggestive of nervous origin—central or reflex. Early morning vomiting before food is taken, accompanied with straining and retching points to chronic gastritis—very often the result

of alcoholism. Severe paroxysms of coughing (as in pertussis) may give rise to vomiting, quite apart from gastric disorder.

Much diagnostic value attaches to the quality of vomit. Though the reaction is almost invariably acid, it is important to ascertain whether the acidity is due to HCl, or to the presence of organic acids, such as lactic acid. The latter points to delay of food in the stomach, especially when accompanied by the presence of food which can be identified as having been taken a long time previously. The presence of mucus in any considerable quantity, points to gastritis ; but the presence of bile is of small significance, as it occurs in persistent vomiting from any cause. Ejection of blood will be dealt with separately. Apart from rare cases of direct communication between the stomach and colon, fæces are not vomited. So-called fæcal vomiting occurs whenever the vomited matter has been retained long enough in the bowel for proteid decomposition to take place, when the colour and odour are those which belong to true fæces.

Marked relief of severe pain by vomiting is suggestive of organic disease of the stomach, especially of ulcer. The pain of gastritis is also relieved in the same way, but not by any means to the same extent ; and in the reflex vomiting set up by biliary or renal colic or appendicitis, the pain is but little relieved or not at all.

6. *Hæmatemesis* or blood-vomiting is characteristic of bleeding into the stomach, only excepting those cases in which blood from the nose, mouth, or air-passages, has been previously swallowed. This may arise from (*a*) external injury ; (*b*) acute congestion due to acute inflammation ; (*c*) mechanical congestion from cirrhosis of the liver, pressure on the portal vein, or long-standing disease of the heart or lungs ; (*d*) erosion of vessels, from ulcer or cancer ; (*e*) disordered state of the blood, as in scurvy, or

yellow fever ; and (f) in rare cases, from the bursting of an aneurism into the stomach.

Small hæmorrhages which are not followed by vomiting may pass unnoticed. In other cases, small quantities of blood in the vomit may be the sole indication that hæmorrhage has occurred. But when the bleeding is sudden and profuse, there is at first a sense of sinking at the epigastrium, sudden pallor, a small frequent pulse, and faintness, which is followed by nausea, and the ejection of somewhat darkened, partly-clotted blood. The next stool contains blackened blood, and for some days after, the motions may be black and "tarry." When the hæmorrhage is scanty and oozing, the blood forms small, dark, granular clots, closely resembling "coffee-grounds." A single attack is seldom directly fatal, though it may give rise to serious and persistent prostration.

Hæmatemesis may be distinguished from hæmoptysis by (a) the darkened colour and acid reaction of the vomited blood ; (b) frequent mixture with food ; (c) the nausea which usually precedes it ; (d) expulsion by vomiting ; (e) the evidence of blood in the subsequent stools ; and (f) by symptoms indicating gastric disorder.

SECONDARY SYMPTOMS.—The secondary disturbances which occur in affections of the stomach are very numerous, but are not of much value for purposes of differential diagnosis. For the most part they arise from nervous derangement, from auto-intoxication, or from inanition.

Digestive System.—The tongue may be variously affected ; thus, it may be red and irritable ; covered with white or yellow fur ; broad, flabby, and pale ; fissured, or patchy. The appetite, though often unaffected, is sometimes increased, but more frequently impaired or lost (*anorexia*). Thirst is a common result of persistent vomiting, or of excessive acidity. The bowels may be costive or act irregularly, and the character of the stools may be altered.

Nervous System.—Weakness, weariness, troubled sleep, frontal headache, depression, irritability, loss of memory, dimness of sight, giddiness, and hypochondriasis are of common occurrence.

Respiratory System.—Dry cough, and asthmatic symptoms.

Circulatory System.—Flushing of the face ; palpitation ; increased frequency, irregularity, or intermission of the heart's action.

Urinary System.—The quantity of the urine may be greatly reduced by persistent vomiting, and by undue retention of the gastric contents. Sometimes it is very acid and deposits urates, uric acid, or oxalate of lime ; while in other cases it becomes unduly alkaline after meals, and deposits amorphous phosphates. In the latter condition, slight albuminuria is not uncommon.

The *general nutrition* may be impaired, as shown by anæmia and emaciation.

FUNCTIONAL DISORDERS OF THE STOMACH.

Normal digestion depends upon the co-operation of three forms of activity—chemical, mechanical, and nervous. The first is related to the gastric secretion, the second to the muscular coat of the stomach, and the third to the nervous system by which both of the former are actuated and controlled. Indigestion or dyspepsia is the result of disturbance in any of these directions ; and thus we meet with secretory, motor, or nervous varieties, according to circumstances. It must be remembered, however, that all functions of the stomach are so closely associated with each other that none can be disturbed in a marked degree, or for long together, without compromising the rest. From a clinical point of view, functional disorders of the stomach may be classed in three groups. First, there are cases in

which the functional disorder is essentially a local affection. Secondly, there are cases in which it occurs secondarily to structural diseases of the stomach. Thirdly, there is a large group in which dyspepsia is dependent upon and subordinate to disease elsewhere than in the stomach—in which it is clearly symptomatic. It is to the first of these groups that the following description applies.

DISORDERS OF SECRETION.

HYDROCHLORIC OVER-ACIDITY, OR GASTRIC IRRITATION, is most frequent in youth and early adult life, and is especially common in those of a nervous, gouty, or rheumatic habit. It may be induced by over-eating, imperfect mastication, by indigestible food, by abuse of alcohol, tea, or tobacco, by nervous excitement, worry, or overwork. It is almost invariably present in cases of gastric ulcer.

The characteristic *symptom* is paroxysmal pain in the epigastrium, which comes on after food. The time at which the pain begins varies according to the quantity and nature of the meal, but it usually begins about two or three hours after food, and reaches a maximum when acidity is at its height, after which it gradually subsides, and disappears when the stomach is empty. (*Cf.* p. 351.) In severe cases the pain may culminate in vomiting, which affords rapid and complete relief. The pain is relieved by alkalies, and by albuminous food. Such attacks may only occur after the chief meal of the day; while, in other instances, they may recur after every meal. The appetite is good, or even excessive; acid eructations are common; and the bowels are costive. The stomach is normal in size as a rule, but from large eating or pyloric spasm, moderate dilatation may occur; fermentation does not take place; and there is no loss of flesh.

There is usually marked tenderness just to the left of the epigastrium, and also at a spot just below and internal

to the left scapula. More or less increase of sensitiveness is often observed over a narrow zone connecting these two points, round the left side. One hour after Ewald's test-meal on a fasting stomach, the contents, when removed by a tube, are found to be abnormally acid (over two per cent.), composed of free and combined HCl. There are no organic acids, or only a trace. The starchy ingredients are badly digested owing to the extreme acidity. The alkaline tide in the urine during digestion is strikingly increased, so that earthy phosphates are often precipitated, causing turbidity of the urine.

It sometimes happens that the acid secretion of the stomach is not confined to the period of digestion, but continues independently of food, and is poured into an otherwise empty stomach (*over-secretion*). In one type of over-secretion there are paroxysms of abdominal pain and vomiting (*gastric crises*), lasting from a few hours to several days, separated by intervals of freedom. They are probably of nervous origin, and are most often met with in *tabes dorsalis*; but they sometimes occur from shock, or in certain cases of visceral displacement such as movable kidney. In a second type, over-secretion is continuous (*gastro-succorrhœa*). Its essential feature is the presence in the fasting stomach at early morning of highly acid active gastric juice. It differs from ordinary retention in the fact that it is found even after the stomach has been emptied and washed out overnight. As a primary affection it is quite rare. More often it is due to pyloric irritation and spasm from pre-existing ulceration; and, in consequence of obstruction to onward passage of the contents of the stomach, progressive dilatation takes place. The symptoms are much the same as those of ordinary over-acidity, but are much more severe, and continue through the night, and the vomit consists of almost pure gastric juice. The condition develops gradually,

persists for a variable time, and may thus pass off ; but recurrence is the rule, after an interval of weeks or months. In course of time, symptoms of gastric dilatation develop side by side with those of over-secretion, and may bring about so serious a failure in bodily nutrition as to suggest the existence of cancer of the stomach. But the presence of free HCl in the fluid removed from the stomach by the tube or by vomiting, and its digestive activity, serve to sharply differentiate the two conditions.

Treatment.—The two main indications in over-acidity are nervous rest, and soothing management of the stomach. The amount of rest, and the mode of enforcing it, must obviously vary according to the circumstances of the patient and the severity of the complaint. The diet should be light and nutritious. A milk diet with cereals is best to begin with, and as improvement takes place, lightly cooked egg, vegetable purées, and tender meats may be added. Fats, such as butter, cold boiled bacon, and cream may be taken, but hot fats are apt to disagree. Acid fruits, preserves, pastry, suet-containing puddings, alcohol, and condiments should be avoided. All food should be well masticated, and the patient should rest a while after each meal. The best drink is plain water, which often agrees better when taken hot ; but weak fresh tea and cocoa are generally admissible. In bad cases, continuous warm moist compresses to the epigastrium are very soothing.

Medicinally, alkalies such as bicarbonate of soda and carbonate of magnesia are of most service, and should be given after meals, and in full doses. Combination with tincture of belladonna is often beneficial, as the latter drug inhibits the secretion of acid to a greater or less extent. Hyperæsthesia of the stomach is best overcome by small doses of arsenic. Constipation requires attention, but care must be taken to avoid irritation of the stomach by strong aperients. Saline aperients and enemata are of most

service. Half a teaspoonful of Carlsbad salt may be dissolved in half a tumbler of hot peppermint water, and given three times a day, half an hour before meals.

Gastric crises must be treated on similar principles. Rest in bed is essential; warm compresses should be applied to the epigastrium; and food should be limited to hot milk and Vichy, or some other alkaline water. Sometimes it is beneficial to wash out the stomach with a solution of sodium bicarbonate (gr. x. ad 3j), and follow up with a hypodermic injection of morphia. Gastro-succorrhœa is admittedly very difficult to deal with successfully by medicinal and general treatment; and the experience of the writer is strongly in favour of gastro-jejunosomy.

DEFICIENT HYDROCHLORIC ACIDITY.—A certain degree of sub-acidity of gastric secretion is probably not at all an uncommon result of weakness and physical depression, but it is relatively of little consequence, so long as the food is simple, nutritious, and well divided, and provided the motility of the stomach is fairly good, because intestinal digestion makes good the deficiencies of the stomach. If, however, imperfectly prepared food is passed on into the bowels, it may set up flatulence and diarrhœa, particularly of that kind in which the bowels are moved soon after meals. In another group of cases, sub-acidity depends upon structural changes in the mucosa, as in chronic gastritis or cancer. Here the symptoms are merged in those of the disease with which it is associated. Lastly, there are rare cases in which not only the acidity is diminished, but the secretion of gastric juice, as a whole, is suppressed (*achylia*). Here again even, there may be no symptoms, or only those of moderate indigestion, or of intestinal disturbance.

The treatment is essentially dietetic, the principles of which may be gathered from the foregoing remarks.

MOTOR DISORDERS.

Provided the walls of the stomach are relatively healthy, the effect of pyloric obstruction is to induce increased muscular action on the part of the stomach, with hypertrophy of its muscular coat, which should be regarded as compensating in its nature. We also meet with irregular action in the form of *spasm*, which acquires a special importance when it is confined, as not infrequently happens, to one of the orifices of the stomach. Thus, irritative affections of the œsophagus are accompanied with spasm of the cardia, and reference has already been made to pyloric spasm as a not infrequent result of the severer types of hydrochloric over-acidity. The latter will again come up for notice in connection with pyloric obstruction.

On the other hand, the motor power of the stomach may be diminished, and give rise to what is called *motor insufficiency*, either as the result of exhaustion of the gastric muscle in contending with long-continued obstruction, or of inherent weakness of the gastric muscular coat (*atony*). The invariable effect of motor insufficiency is to delay the passage of the gastric contents through the pylorus into the bowel, and all the symptoms to which it gives rise are attributable to this fact. Such cases admit of two-fold division according to the degree of delay. In the one group, favoured by the long interval of fasting and the recumbent posture of the patient, the stomach becomes completely emptied during the night, so that a fair start is made at the beginning of each day. In the other group, this does not occur. The stomach is never empty, fermentation takes place, and the stomach becomes somewhat dilated. The former is sometimes called "stagnation," as distinguished from the latter, which is termed "retention." Stagnation is a common result of atony of the stomach, but retention seldom occurs apart from pyloric obstruction.

Atony of the stomach is a term which is clinically applied to functional weakness of the muscular coat of the stomach occurring independently of chronic pyloric obstruction, though the distinction is not pathologically justified. It is one of the commonest forms of dyspepsia. It may be due to local causes such as habitual over-eating and drinking, especially in connection with hurried meals. But more often it is secondary to nervous depression, physical or mental fatigue, hysteria, anæmia, or general debility; or it occurs as a sequel of exhausting fevers. An acute and serious form of atony, though fortunately of exceptional occurrence, may result from local shock or injury, as after a fall on the back, or abdominal operations.

The subjects of atony are more often pale, thin, and nervous; but are sometimes stout, plethoric, and subject to gouty or rheumatic troubles. The appetite is generally poor, and food is followed by more or less persistent weight and fullness at the epigastrium, which are related more to the bulk than the character of the food, and usually worse towards the end of the day. Sometimes the discomfort is more acute, and radiates into the left side or back. Flatulent eructations are frequent, and often very persistent; but regurgitations are rare. Constipation is almost invariable. The general condition is characterised by headache, lassitude, and a feeling of being always tired.

On palpation of the stomach, splashing and gurgling sounds are readily produced, often during the whole of the digestive period. Moderate dilatation of the stomach is the rule, with stagnation of contents, and the lower curvature frequently reaches to the level of the umbilicus or sometimes beyond; but actual retention is rare. In many cases also, especially in women, there is some downward displacement of the stomach, and not infrequently of one or both kidneys as well. The secretory behaviour of the stomach presents no constant features.

Treatment.—All sources of fatigue and depression should be dealt with as far as possible, and rest plays an important part in the treatment of atony. In certain cases of nervous origin a course of Weir-Mitchell treatment may be of great value. With regard to diet, the chief aim is to prevent undue distension of the stomach, and yet at the same time to maintain as high a standard of nutrition as possible. For such reasons, the diet should be compact and nutritious; bulky vegetables and easily fermentable articles should be avoided, and the amount of fluid taken *with* meals should be closely restricted. Milk diet is objectionable except in cases kept to bed. The recumbent posture greatly facilitates the emptying of the stomach; and, on this account, rest is desirable after meals, and especially after fluids. The best times for taking fluids are :—an hour before rising in the morning; at teatime (without solids, except perhaps, one or two plain biscuits); and at bedtime. The patient should lie down after tea, if possible, for half an hour to one hour. Abdominal massage and exercises to strengthen the abdominal walls are helpful, and great comfort is derived from a light, well-fitting abdominal support, *without* corsets. Very active aperients should be avoided; and as a rule it is better to rely upon massage, and occasional enemata. Among drugs, the most useful is strychnia.

Dilatation of the stomach is a common anatomical expression of motor insufficiency. In a moderate degree it is a common result of flatulent distension and stagnation of the stomach contents arising from atony; but marked dilatation rarely occurs apart from pyloric obstruction of some kind associated with retention. The circumstances under which it arises are exactly analogous to those which give rise to dilatation of the heart, namely, strain brought to bear upon a stomach which has lost its tonicity. In previous editions of this work, a separate section has

been allotted to dilatation, but the symptoms with which it is so frequently associated are in reality not directly dependent upon dilatation as such, but upon motor insufficiency which cannot be practically separated from the atony or pyloric obstruction on which insufficiency depends, and therefore is more conveniently considered under these headings. The physical signs of dilatation are prominence of the left upper part of the abdomen with relative depression of the epigastrium, extension of the area of the stomach or percussion, persistent splashing sounds on succession, and experimental proof by artificial inflation. Another feature, which is characteristic, is increase in the amount of fluid obtained by tubage after a test-meal.

SENSORY DISORDERS.

DISTURBANCES OF APPETITE.—A morbid craving for food in excess of the needs of nutrition is known as *bulimia*. It often occurs in connection with over-acidity, but it is also met with as an independent affection in hysterical and nervous subjects. Another disorder of the appetite is characterised by a special craving for certain articles of food, or even for non-alimentary substances (*parorexia*). It is most common in pregnancy, and in nervous chlorotic girls. Loss of appetite (*anorexia*), as a symptomatic condition is familiar enough; and insane persons often persistently refuse food. But cases are sometimes met with in nervous subjects—chiefly in young women—independently of any other symptom, in which, for some obscure motive, food is systematically refused (*anorexia nervosa*). Steadily progressive inanition is the obvious result. Isolation of the patient under gentle but firm control, absolute rest in bed, resolute feeding, massage and electricity—with patience—usually suffice to cure.

GASTRIC HYPERÆSTHESIA. GASTRALGIA.—*Gastric hyper-*

æsthesia is characterised by a morbid sensitiveness of the stomach in the presence of food, giving rise to pain and sometimes to vomiting. The pain varies widely in degree and duration, and is also very irregular in its relation to particular foods. It may be worse after liquids than solids. The appetite is little affected, and may be exaggerated; and the bowels are constipated. Hyperæsthesia is frequently associated with other forms of gastric disturbance of an irritative type. It is also of common occurrence in anæmia and nervous functional disorders, especially in women; or it may be connected with abuse of tea, coffee, alcohol, or tobacco.

Gastralgia is a neuralgic affection, characterised by irregular paroxysmal attacks of gastric pain, separated by intervals of freedom, with no definite relation to food, and unaccompanied with other obvious local disorder or structural change. Thus restricted, gastralgia is not a frequent affection. It is more common in women than in men. Nervous over-strain, hysteria, anæmia, gout, malaria, lead-poisoning, and sexual excess may be mentioned as possible causes.

The affection is characterised by paroxysms of pain, varying much in severity, beginning suddenly, with no constant relation to food or to any particular phase of digestion, and lasting from a few minutes to many hours, extending it may be over some days, and then passing off, to recur at widely irregular intervals. In mild cases the pain is strictly confined to the stomach; while in severe cases it radiates thence in various directions. It is not relieved by food, nor by alkalies. If it comes on with a full stomach it may end suddenly with vomiting; but if the stomach is empty, or nearly so, it more often terminates gradually, and is attended with a copious discharge of pale limpid and feebly acid urine. During the intervals between the paroxysms, digestion is natural and painless.

Gastralgia needs to be carefully distinguished from symptomatic gastric pain, intestinal colic, biliary and renal colic, and intercostal neuralgia.

In the *treatment* of painful neuroses of the stomach, rest in the fullest sense, is essential. Probable causes should be carefully investigated, and dealt with as far as possible. The diet should be bland in quality, but at the same time as nourishing as possible. If left to themselves, patients are apt to take far too little nourishment, and with tact and careful selection of food the quantity can often be largely increased, without more distress than when less is taken. Alcohol, tea, coffee, and tobacco-smoking should be forbidden. Locally, continuous application to the abdomen of warm, moist compresses is useful. Medicinally, for hyperæsthesia sodium bicarbonate in combination with arsenic before meals is of value, and in some cases nitrate of silver may be given with advantage. In severe gastralgia, phenazone in combination with bromide of potassium may be recommended, or, failing this, a hypodermic injection of morphia should be given.

GASTRITIS.

ACUTE GASTRITIS, in a large majority of cases, depends upon damage arising from errors in diet, abuse of alcohol, and the products of fermentation (*simple gastritis*). In other cases it occurs as a local manifestation of specific fevers (*infective gastritis*); while in a third group it is due to corrosive poisons (*toxic gastritis*).

The *anatomical changes* vary from a simple catarrhal inflammation of the gastric mucous membrane, chiefly affecting its glandular structures and attended with excessive production of mucus, up to severe ulcerative lesions attended with hæmorrhage.

The *symptoms* vary widely in degree according to

cause. In mild cases the condition is ushered in with more or less pain and tenderness at the epigastrium, flatulent eructations, complete anorexia, nausea, and some thirst. The temperature may be normal or slightly raised. In other cases, however, the onset is more abrupt and painful, and is attended with a rapid rise of temperature, sometimes to a high grade. The tongue is thickly coated, and often indented by the teeth; taste is perverted, and the breath is offensive. Acrid regurgitations are not uncommon. There is considerable malaise, with frontal headache and general prostration. Vomiting is rarely absent, and may be incessant. The vomit at first consists of imperfectly digested food imbedded in mucus, and afterwards of bile-stained mucus sometimes streaked with blood, which is brought up with great effort, and accompanied with distressing nausea and retching. It contains, as a rule, abundant organic acids, but no free HCl. In adults the bowels are more often confined, but diarrhoea is the rule with children. The urine is scanty, high-coloured, and deposits urates. Under proper treatment, simple acute gastritis usually subsides in a few days, but the stomach is apt to be rather sensitive to disturbance for some time afterwards. In neglected cases, it may pass into the chronic form.

In severe cases of the toxic type, the symptoms are much more alarming. The pain is intense; the abdomen is tumid and very tender; swallowing is often difficult and painful; and there is constant vomiting of mucus, blood, and sometimes shreds of mucous membrane. Albuminuria is not uncommon. In the worst cases, fatal collapse may set in; and in those which recover from the immediate attack, serious results such as stricture of the gullet, or chronic gastric troubles may follow, under which the patient ultimately sinks.

Treatment.—The primary indication is rest for the

stomach. Nothing should be given for the first day but small quantities of some simple drink as rice-water or toast-water ; or, if there be much prostration, sips of iced champagne or brandy and soda water. On the second day hot milk and Vichy water, milk and barley water, or thin broth with plasmon will generally be well borne. From this point a gradual return may be made to more nutritious food. With careful dieting on these lines little medicinal treatment is required, except for the relief of specially troublesome symptoms. After the initial vomiting has ceased, a dose of calomel is often useful, especially when the bowels are confined. For troublesome eructations, or persistent vomiting, a mixture containing sodium and magnesium carbonates in peppermint water may be given, with ice to suck, and a mustard plaster to the epigastrium ; or in some cases it may be well to wash out the stomach by means of a tube. For the relief of severe pain, warm compresses should be applied to the epigastrium, or a hypodermic injection of morphia may be justifiable. When the acute symptoms have subsided, hydrochloric acid and strychnia are advised. (For the treatment in detail of toxic gastritis the reader is referred to special text-books.)

CHRONIC GASTRITIS may occur as the sequel of the acute form, but more commonly it arises from frequently recurring irritation by improper or too abundant food, from abuse of alcohol or tobacco, from imperfect mastication, or from chronic venous congestion. As a complication of functional and organic disease of the stomach, it is extremely common.

Anatomical Changes.—In general terms the inflammatory process is characterised by early irritation, followed by proliferation, desquamation, and degeneration of the mucous and glandular epithelium, associated with cell-infiltration of the sub-mucosa. The mucosa becomes thickened and covered with a layer of sticky mucus. The

surface is often irregular, and (in later stages) acquires a dull brownish or slate-grey colour. Marked variations in distribution, intensity, and mode of evolution are observed in different cases. In some the mischief is chiefly superficial; in others, it falls mainly upon the gastric glands; while in a third series, interstitial infiltration and overgrowth are the most prominent features. Doubtless the variable clinical manifestations of chronic gastritis are in some degree related to such differences, but our present knowledge does not suffice for definite classification on an anatomical basis. As regards ulterior results, a certain number of cases recover under treatment; in others progressive atrophy of the mucosa and its glands takes place; while in a few cases fibroid thickening takes place, which, being generally most marked at the pylorus, may cause obstructive narrowing of that orifice. The muscular coat is not usually involved, at any rate not until at quite a late stage.

The *symptoms* of this affection are by no means characteristic. Essentially we have to deal with a process of chronic dyspepsia, which is due to progressive secretory insufficiency on the part of the stomach, and not to motor failure. In the earlier stages, the secretion of mucus is increased, and sometimes the index of HCl acidity may be temporarily raised: but, as destructive changes proceed and atrophy takes place, both acidity and the quantity of mucus steadily decline.

The onset is insidious. The earliest symptom is generally loss of appetite, with a preference for stimulating and piquant dishes. When there is any appetite, a few mouthfuls will often satisfy it. The tongue is coated and indented, especially when there is much fermentation, taste is perverted, and the breath is offensive. Another early symptom is epigastric fullness and discomfort, which is worse after meals, especially after solid food. Some

diffused epigastric tenderness is generally observed. Flattulent eructations—sometimes offensive and acrid—are of common occurrence. Nausea and vomiting are not infrequent, especially that form known as “morning vomiting.” Vomiting after meals returns the food partially digested, and imbedded in a quantity of sticky mucus. In the later atrophic stage the amount of mucus is much less. Organic acids are generally present in considerable amount, but general HCl acidity is most often deficient and free HCl is absent. Occasionally the vomit is streaked with blood. Constipation is the rule; and the urine is scanty, high-coloured, and deposits urates. Nutrition is not affected at first; but in the later stages there may be so much emaciation and weakness as to suggest the existence of malignant disease. The nervous system is depressed, headache is common, and there is usually a marked inaptitude for physical or mental exertion.

The progress is slow and tedious, but many cases recover or greatly improve under treatment, if taken in hand early.

The *diagnosis* requires care. The most characteristic signs are (in the stage at which the affection usually comes under notice) deficiency of HCl with absence of free HCl, corresponding deficiency of pepsin, and excess of mucus. The last two points serve to distinguish chronic gastritis from simple functional deficiency of HCl. The diagnosis further turns upon the confidence with which ulcer and cancer of the stomach can be excluded. It is in cases attended with thickening and obstruction of the pylorus, and motor insufficiency, that difficulty chiefly arises.

As to *treatment*, bland, simple, well-divided food is essential. Milk foods and broths or beef-tea, fortified with milk, plasmon, eggs, or pounded meat, usually agree. In milder cases light meats free from fat, vegetables, and milk puddings may be given. Vegetables are more acceptable when pulped and rubbed through a sieve. Except as a zest

to appetite in selected cases, alcohol should be avoided. A glass of hot water should be sipped at early morning (with the addition of an aperient saline, if the bowels are not acting well), and repeated an hour before dinner, and again at bedtime. When there is much fermentation, and especially if there is evidence of retention and dilatation, washing out of the stomach should be resorted to. The importance of exercise, fresh air, with change of scene and occupation, should be kept in mind. The most useful medicines are those which reinforce the weakened digestive secretions—namely, dilute hydrochloric acid and pepsin. Osler also speaks well of pancreatin and soda (gr. x.-xv. of each, after meals). Nitrate of silver (to be given with care) is worthy of trial in troublesome cases.

ULCER OF THE STOMACH.

The simple or peptic ulcer of the stomach is generally thought to be due to the destructive action of gastric juice upon localised areas of the mucous membrane, the resistance of which has been impaired or arrested by local injury, sub-mucous extravasation or thrombosis, or localised bacterial invasion; but there are many points which yet remain to be cleared up. The rapidity of the ulcerative process varies widely, and acute and chronic types are described, but the acute form cannot be distinguished clinically until after the event. Ulcer is very rare in children, and is most frequent among females between 20 and 30 years of age, and among males between 40 and 50.

ANATOMICAL APPEARANCES.—A simple gastric ulcer is generally round or oval, from one-fourth to one-half an inch diameter, with thin clean-cut margins, deepest in the centre like a shallow funnel, but varying in depth from mere destruction of the mucous membrane to complete

perforation. It is more often single, and situated on the posterior wall near the pyloric end. Erosion of important vessels may occur, leading to serious hæmorrhage. In chronic cases the ulcers are larger and more irregular in shape, with indurated and thickened edges, and are apt to be attended with peritoneal adhesion of the stomach to adjacent parts, especially the left lobe of the liver, and the pancreas. The ulcer may cicatrise, with or without shrinking and puckering of the mucous membrane; or perforation may occur, setting up either a general peritonitis, or local suppuration; or ulceration may extend into adjacent organs to which the stomach has become adherent. Pyloric obstruction may result from ulcer, either as the result of pyloric spasm or cicatricial contraction. Ulcer is almost always associated with hydrochloric over-acidity, which may be confined to the periods of digestion, or continuous (*gastro-succorrhœa*).

SYMPTOMS.—The clinical history is very variable, owing to conditions which are difficult to define. Sometimes the disease remains quite latent until the sudden occurrence of hæmorrhage or perforation; in other cases there are only the signs of over-acidity without obvious hæmorrhage; but more often we meet with a group of symptoms, which, taken in the aggregate, are characteristic of gastric ulcer—namely, pain, tenderness, vomiting, and hæmatemesis.

Of these *pain* is the most constant; but it varies greatly in intensity and character. Different kinds of pain may be distinguished. First, an epigastric pain with more or less deep tenderness, for the most part strictly localised to a small area, coming on immediately after food, and striking through to the back. It is probably directly due to the ulcer itself. Then again, there is, very commonly, a pain with superficial tenderness referred to a lateral zone on the left side, with maximum points of intensity to the left of the epigastrium, and in the back below and inside

the angle of the left scapula. It is probably due to gastric irritation from over-acidity, reflected through the spinal cord into the nerves of the abdominal wall. More rarely we meet with colicky pains, relieved by pressure, probably associated with pyloric spasm; and in certain old cases there may be dull dragging pains from inflammatory adhesions. But whatever the precise explanation of pain in gastric ulcer, it begins as a rule immediately after food, and increases as digestion proceeds, until the stomach is finally emptied by vomiting or the passing on of its contents into the bowel. Although there is doubtless some general relation between the seat of pain and the position of the ulcer, it is not precise enough to justify any trustworthy conclusions. *Tenderness on pressure* is almost constantly present, especially the superficial tenderness just to the left of the epigastrium and in the back; which, if not due to hydrochloric over-acidity, is at any rate indistinguishable from it. Its localisation is the same, whatever position the ulcer may occupy in the stomach. *Vomiting* is another frequent symptom. It usually occurs easily and without nausea about two hours after food, at the height of digestive acidity, and is followed by the immediate relief of the pain. *Gastric hæmorrhage*, though it only occurs in less than half of all cases, is the most characteristic symptom of ulcer. It is usually profuse, and promptly followed by vomiting (*hæmatemesis*); but it may occur without vomiting, and under these conditions, it can only be detected by the discovery of blood in the stools. The quantity may be so small, that chemical tests are necessary for its detection (*occult bleeding*).

The appetite is maintained or increased, but the patient is afraid to eat; and—partly from inanition, and partly from hæmorrhage—profound anæmia and malnutrition may develop and become exceedingly serious.

Perforation occurs in 5 to 10 per cent. of all cases. It

is usually characterised by the sudden occurrence of severe abdominal pain, attended by collapse, low surface-temperature, feeble pulse, rapid shallow breathing, and generally by vomiting. Unless the patient dies in the meantime, peritonitis supervenes; and, unless relieved by prompt operation, the patient usually dies within forty-eight hours. If protective adhesions are present, perforation may occur without escape of the gastric contents into the peritoneum.

The duration of gastric ulcer varies greatly. The disease usually terminates in one of three ways:—gradual recovery by cicatrisation, which is the rule; rapid death by perforation or hæmorrhage; gradual death from exhaustion and malnutrition. More rarely, troublesome sequelæ may occur, of which sub-diaphragmatic abscess is the most important. Gastric ulcer has to be distinguished from gastralgia, gastric over-acidity, cancer of the stomach, and gall-stones, and in some cases diagnosis presents great difficulty. In such cases careful examination of the fæces for evidence of occult bleeding may be of the greatest value. (Refer to Appendix.)

TREATMENT.—Diet ranks first in importance. Apart from recent hæmorrhage, no advantage is gained by abstaining from all food by the mouth. Plain milk, with the addition of citrate of soda—grs. ij. to the ounce—is best to begin with, in ten daily doses of two ounces every two hours. This quantity should be gradually increased to four ounces; by which time cereals and soft-boiled eggs may be addêd. No meat foods should be taken for the first four weeks, and great care should be exercised in the diet for at least another month. For *pain*, hot fomentations to the abdomen are of service, and (when specially severe) hypodermic injections of morphia. Carbonate of bismuth combined with tincture of belladonna, or nitrate of silver (gr. $\frac{1}{4}$ doses) in solution, are useful medicines; and a

teaspoonful of olive oil half an hour before food has often a most soothing effect on the stomach. When vomiting is severe, feeding by the mouth should be suspended for a day or two, and nutrient enemata substituted. *To empty the stomach of acid products of digestion*, saline aperient waters should be periodically exhibited. For *hæmorrhage*, everything by the mouth should be withheld for three days, except a little ice to suck. A nutrient enema, consisting of the yolks of two eggs, a teaspoonful of grape-sugar, ten ounces of peptonised milk, and a good pinch of salt, should be given every eight hours. If not retained, tincture of opium should be added to it, which has the additional advantage of relieving restlessness. Ordinary styptic medicines are of no value, but an occasional dose of $\mathfrak{M}$ 20-30 of adrenalin chloride (1 in 1000) may be useful. Enemata of normal saline may be needed to relieve thirst, to cleanse the bowel while nutrient injections are being used, and to correct constipation.

DUODENAL ULCER.—Ulceration, exactly similar to that which has just been described as affecting the stomach, may occur in the duodenum, either as an independent condition, or in association with gastric ulcer. The ulcer is commonly situated in the first part of the duodenum, in fairly close proximity to the pylorus. It is characterised by gnawing pain about midway between the navel and xiphoid cartilage, coming on from three or four hours after a meal; and sometimes so severe as to simulate an attack of biliary colic. Coming on thus at a time when the patient is beginning to feel hungry again, the pain has been described as "hunger-pain." It is apt to strike through to the back when severe, and round to the right side. It is relieved by food, or an alkaline drink. It is accompanied very frequently by a sense of distension, and flatulent eructations; but actual vomiting is unusual. In many cases, hæmorrhage takes place, which is revealed either by

obvious melæna, or by the discovery of "occult" blood in the stools. There is generally some tenderness on pressure over the region of the duodenum.

After a variable time, measured by days, weeks, or longer, the attack may pass off; but it is liable to recur. During the intervals the patient may feel perfectly well. It may terminate in recovery, or the strength may be gradually worn out by frequent recurrence; or (more rarely) fatal hæmorrhage or perforation may take place. The treatment is the same as for gastric ulcer.

CANCER OF THE STOMACH.

The stomach is one of the most frequent seats of cancer. Hereditary tendency, advanced life, and chronic irritation may be predisposing causes. It occasionally supervenes on chronic ulcer. It is more common in the male sex.

ANATOMICAL APPEARANCES.—Cancer more often involves the orifices of the stomach, especially the pylorus; and is almost always primary. *Scirrhus*, or hard cancer, is the commonest type. In other cases, it is of the soft or *encephaloid* type; whilst a third type is characterised by cylindrical cells, arranged in irregular tubules, imbedded in a tolerably abundant stroma, and known as *adeno-carcinoma*. Either of the last two may be attended with mucus formation (*colloid cancer*?). Sooner or later the growth breaks down in parts, leading to irregular ulceration, which sometimes goes on to actual perforation. It is apt to involve neighbouring organs and structures, either by direct extension, or as a secondary deposit.

The stomach is variously affected, according to the seat and development of the growth. When the pylorus is affected, the orifice is obstructed, and the stomach becomes dilated, while if the cardia alone be affected, the œsophagus dilates above, and the stomach is small.

SYMPTOMS.—The onset is usually gradual. At first the symptoms are not easy to distinguish from those of chronic gastritis; but dyspepsia with complete loss of appetite coming on in the later life of one who has hitherto been free from such trouble, should always arouse suspicion. Later on, there is more or less constant *pain* at the epigastrium, gnawing or lancinating, and striking through to the back and shoulders. It is aggravated by food, and relieved by vomiting, but not so completely as in ulcer. *Tenderness* is almost invariable. *Vomiting* is seldom absent, and is induced partly by commencing pyloric obstruction and partly by irritation set up from fermentation. As obstruction increases, and dilatation of the stomach takes place, vomiting becomes less frequent, but the vomit is more abundant, frothy, and may contain *sarcinæ*. Free hydrochloric acid is absent, or is much reduced, while lactic acid is uniformly present. With obstruction of the cardia, food is returned almost immediately, and but little altered. *Hæmatemesis*, as such, occurs in less than half of all cases.

As the hæmorrhage is generally gradual and scanty, it becomes coagulated in fragments, blackened by the action of the gastric fluids, giving rise to a “coffee-ground” appearance in the vomit. Evidence of blood in the stools (*melæna*) may generally be detected either by the naked eye, or by chemical test even in the absence of hæmatemesis. The general health deteriorates from the first in a steadily progressive manner, and often with rapidity. Pallor, emaciation, increasing weakness, with a peculiar expression of anxiety, are the most striking features.

In a majority of cases a palpable *tumour* in the epigastric region can sooner or later be made out, but in others nothing of the kind can be detected from first to last. Its exact position and character vary in different cases. For the

most part it is hard, tender to touch, and irregular in shape. Though sometimes fixed, it is more often movable, especially when affecting the pylorus; and the tumour may often be seen to rise and sink with respiration. Secondary growths in the liver may also be felt, and may sometimes be the only discoverable indication of local structural disease. The signs of pyloric obstruction and gastric dilatation are also very commonly observed.

The average *duration* of the disease is about one year, and it is always fatal. Death occurs from exhaustion, and is sometimes preceded by femoral thrombosis.

DIAGNOSIS.—The chief points which serve to distinguish *cancer* from *ulcer* of the stomach are—rare occurrence except in later life; greater persistency of the pain; more diffused tenderness; loss of appetite; recurrent but scanty hæmatemesis; frequent evidence of persistent occult bleeding on examination of the stools; absence of free HCl in the gastric contents; the signs of dilatation; early deterioration of general health; the frequent presence of a palpable tumour; and finally a history of steadily progressive trouble from the onset of symptoms.

TREATMENT can only be palliative, apart from surgical measures. The diet should be light, but nutritious; meaty foods may be given in moderation, on condition that they are very finely divided. Vegetables should be pulped, or given as purées. With evidence of retention, systematic lavage is of great value. Medicines are of little use, but hydrochloric acid with pepsin and strychnia after food is sometimes helpful. Surgical interference is rightly attracting increased attention: (*a*) exploratory, for early diagnosis; (*b*) gastro-jejunostomy, as a palliative; and (*c*) gastrectomy, for removal of growth where practicable.

PYLORIC OBSTRUCTION.

Pyloric obstruction may arise in various ways. In the first place, it may be due to functional spasm of the pyloric muscle, occurring usually in connection with pyloric ulcer. It is characterised by attacks of severe pain, with violent eructations or actual vomiting, coming on at the height of digestion. At first the attacks only occur at intervals, but afterwards become more frequent, until they take place whenever food is taken. In a second group of cases, obstruction is due to organic but benign causes, such as cicatricial contraction following upon peptic ulcer or toxic gastritis; or to adhesions or constricting bands from old peritonitis. In a third and largest group, obstruction depends upon malignant growth.

The leading feature of pyloric obstruction is gastric dilatation, the principal signs and symptoms of which have already been described. It will be remembered that dilatation is also a common result of atony of the stomach; but, in atony, the history is uneventful. The course is slow, the dilatation is moderate, the symptoms are usually limited to simple discomfort and flatulence without pain or tenderness; vomiting is rare, there is no visible peristalsis, and no tumour. In dilatation from pyloric obstruction, on the other hand, there is often a history of previous ulcer, hæmorrhage, or severe inflammatory conditions; the course (taken as a whole) is progressive, and often rapid; dilatation is marked; actual pain (often severe) and tenderness are the rule; vomiting is frequent and copious; and a tumour, or tumour-like resistance is frequently found.

The extent of gastric dilatation is largely in direct proportion to the degree and duration of the obstruction at the pylorus; but much also depends upon the cause of the obstruction, together with the condition and resistance of

the gastric wall. Thus, in cases of cicatricial contraction after ulcer, with a relatively normal condition of the muscular coat, the obstruction may be overcome for a time by hypertrophy of the stomach wall. But as obstruction continues and increases, the stomach becomes exhausted, and motor insufficiency supervenes with dilatation. On the other hand, in cancer with chronic gastritis, motor insufficiency and dilatation occur early. The results of tubage of the stomach after a test-meal will show that delay of digestion is in proportion to the amount of insufficiency. The fasting stomach at early morning may at first be empty; but, sooner or later, there will be evidence of actual retention, remains of food taken on the preceding day will be found, together with all the signs of intra-gastric fermentation.

The existence of pyloric obstruction having been established, the next step is to discover its cause; and, in this connection, the most important question is as to whether the condition is benign or malignant in its nature. The chief considerations in favour of malignant disease (cancer) are the following. The age of the patient is generally over 40 years. It is rare to meet with any history of previous ulcer, toxic gastritis, or likely causes of local peritonitis. On the other hand, a history of good digestion up to the present illness is very suggestive. The course is uniformly progressive from the first. There is steady loss of flesh and strength, and the loss of appetite is complete. The pain is more continuous than in benign conditions and less severe, as a rule. Visible peristalsis is less common. The vomit shows deficiency of HCl acidity, and complete absence of free HCl in all but a very small minority of cases. A palpable tumour is more common, and it is generally hard and tender on pressure.

The *treatment* is that of the cause on which the obstruction depends.

SUB-SECTION IV.

DISEASES OF THE INTESTINES.

Before proceeding to describe the diseases proper to the intestines, four important symptomatic conditions must be briefly discussed.

I. DIARRHŒA.

Diarrhœa is proximately due either to unusual fluidity of the intestinal contents, or to increased peristaltic activity of the intestinal walls, or to both of these conditions combined. These results may be brought about by intestinal irritation, especially by errors of diet; by inflammatory or structural changes in the intestinal wall; by toxic conditions such as uræmia and infective disease; or by disturbance of innervation.

The *characters* of the stools vary greatly according to circumstances. They may be (a) *fæculent*, or contain no true fæcal matter; (b) *brown, green, or yellow*, due to various alterations of bile pigment; (c) *slimy*, due to admixture with mucus; (d) *serous*; (e) *bloody*; or (f) *lienteric*, containing scarcely altered food. They may be alkaline or acid; in the latter case they are very apt to irritate the anus and surrounding parts. They may be passed easily, with straining (*tenesmus*), or with griping pains (*tormina*). Diarrhœa may also be associated with various digestive disturbances. If it continues for any considerable period it is attended with debility, wasting, and prostration. It may be added that diarrhœa with slimy stools always implies an inflammatory condition of the bowels; and when accompanied with *tenesmus*, the lower or larger intestine is always involved.

The main principles of *treatment* are to remove all sources

of local irritation, and to soothe any subsequent irritability of the bowel.

In all cases the *diet* requires careful attention. In some acute cases it is desirable to suspend all ordinary nourishment, and to give only sips of toast-, barley-, rice-, or albumenised-water for the first 24 or 48 hours. Otherwise the diet should be limited to light liquid foods, of which milk preparations are the best. No solid foods should be given until the bowels are fairly steady again.

Medicinally, the first object is to get rid of irritating material from the bowel. This is often accomplished by the diarrhœa itself; but sometimes, a mild aperient such as castor oil is desirable, or the irrigation of the colon by injections of warm water. In mild cases, nothing more may be needed; but more often further treatment is necessary to soothe the irritated bowel. For this purpose, chalk and bismuth preparations are particularly useful, exhibited with some carminative water. In severe cases, an opiate may be added, such as paregoric or Dover's powder, but never as a matter of routine. Small doses of sub-acetate of lead, or sulphate of copper are sometimes given, but astringent drugs have a much more limited application. On the other hand, intestinal antiseptics, such as small doses of calomel or hyd. *ē. creta*, bismuth salicylate, or salol may be of great service. In diarrhœa dependent on nervous disturbances, treatment should be directed rather to the general nervous system, with careful regulation of the diet. Diarrhœa due to affection of the colon sometimes demands local treatment, in the shape of irrigation, or injections medicated with borax (antiseptic), opiates (sedative), or nitrate of silver in very dilute solution (astringent).

II. CONSTIPATION.

Constipation may arise from various causes, among which

the following are the chief :—habitual neglect of the calls of nature, sluggish and sedentary habits, weakness of abdominal walls and enteroptosis, insufficient or unsuitable food, chronic catarrh and congestive conditions, abuse of aperients, neurasthenia and various nervous disorders, intoxications such as plumbism, and local forms of obstruction.

The earliest stage of almost all forms of chronic constipation, apart from certain neurasthenic and hysterical cases, may be described as one of atony of the colon. The usual symptoms are general lassitude and depression, headache, loss of appetite, with a coated tongue ; but the amount of disturbance varies widely in different persons. As time goes on, various secondary effects may be observed, such as an abdominal tumour from fæcal accumulation, piles, and colicky pains. In certain cases, retained saybalæ may set up a catarrhal or an ulcerative type of colitis, causing diarrhœa. In others, a spastic condition of the colon is developed, especially in neurotic subjects ; and with many of the latter, constipation is of a spastic rather than of an atonic type throughout.

Treatment.—In ordinary chronic constipation, the diet first requires attention, and such articles as oatmeal, vegetables, bran bread, and fruits are usually of service. A liberal amount of fluids should be taken. Exercise of all kinds is indicated so long as it stops short of fatigue ; and, in aggravated cases, systematic abdominal massage is a valuable resource. Great discretion is needed in the use of aperients, and much harm is done by their indiscriminate employment. As a rule it is better to give minimal doses at regular short intervals, than large occasional doses. In many cases aloes, in combination with nux vomica and belladonna, is very useful, or cascara sagrada. When associated with portal congestion, saline aperients are preferable, taken with hot water before breakfast, while some-

times it may be sufficient to introduce a glycerine suppository each morning. Above all, the *habit of regularity* should be cultivated in every possible way.

Spastic constipation requires a soothing plan of treatment. Rest in bed, and freedom from worry and strain are of the first importance. The diet should be bland but thoroughly nutritious. Neurasthenic patients are apt to take far too little food. Coarse food, potatoes, and cabbage are undesirable, and all vegetables should be given in the form of purées. The writer has seen great benefit from the continued use of warm moist compresses to the abdomen, and a well-fitting abdominal support is often of great service after the patient leaves her bed. Massage of the abdomen should be avoided. Locally, half a pint of warm olive oil should be injected slowly into the rectum every night; and later on less frequently. As to drugs, belladonna and carminatives are of most value. Opiates should be avoided.

III. MELÆNA.

Melæna, or the discharge of blood per rectum, as already pointed out, always follows hæmorrhage into the stomach. But it may also be due to intestinal hæmorrhage, arising from causes similar to those mentioned when describing hæmatemesis. When the bleeding is slight, comes from the small intestine, and is discharged slowly, the blood becomes altered, and the fæces are uniformly blackened or "tarry." If from the same source, but copious and quickly removed, the tarry change is not observed. When coming from the rectum or the anus it is bright and unaltered, and *not* uniformly mixed with the fæces. By attention to these points, and to the symptoms and signs associated with them, the origin and cause may usually be determined.

The special *treatment* of melæna must be conducted on

the same principles as that of hæmatemesis. Oil of turpentine is especially useful. Fissures, ulcers at the anus, or hæmorrhoids require surgical interference.

IV. COLIC.

Colic or painful spasmodic contractions of the bowels may be due to irritation of the bowel by improper or undigested food, flatulent distension, constipation, or worms;—by inflammatory or structural disease of the bowel; by toxic conditions, such as lead poisoning; or by nervous disturbance—central or reflex.

Symptoms.—The most characteristic feature of colic consists of paroxysmal griping *pains*, which generally begin about the navel and spread thence over the belly. The pain doubles up the patient, and is relieved by firm pressure. The belly is either hard and retracted, or tense and distended; and the bowels, though usually constipated, are sometimes relaxed. The attack may be associated with faintness, nausea, or vomiting, but there is no pyrexia. After a variable duration, the attack gradually subsides, but considerable abdominal soreness may persist for some time. Care should be taken to distinguish colic from acute intestinal strangulation, and from perforation of the stomach or bowel.

Treatment.—As a rule, it is well to begin with an enema, and follow it up with a purgative, such as castor oil, to which a little laudanum may be added with advantage. To relieve pain and spasm, belladonna, with diffusible stimulants, such as spirits of chloroform, or tincture of cardamoms, may be given; or morphia may be subcutaneously injected. Hot fomentations or turpentine stupes applied to the abdomen are useful adjuvants. The general conditions on which the colic depends subsequently require attention.

INTESTINAL INFLAMMATION.

ENTERITIS.

Enteritis may be caused by errors in diet, by decomposing food, and other toxic matters, by exposure to cold, or by anything which irritates the bowel. Enteritis also occurs as a symptomatic condition in many infective diseases, in connection with portal congestion, or by the extension of inflammation from adjacent parts. It may be either acute or chronic.

In acute cases the mucous membrane (as seen *post mortem*) is sodden, and covered with mucus. The lymph follicles are almost always swollen, especially in children, and sometimes exhibit small central ulcers (follicular ulcer). In chronic inflammation, the mucosa is firmer and somewhat thickened, and it may lead to degeneration and atrophy of the glandular structures. Intractable catarrhal ulcers may occur as the combined result of desquamation and mechanical injury.

The *symptoms* of primary acute enteritis vary according to its seat and intensity. In cases of moderate severity the disorder usually begins abruptly with diarrhoea, vomiting, and colicky pains. The stools at the outset are of a faecal nature, brown, and offensive; but afterwards become paler or green-coloured, and consist chiefly of intestinal secretions, and mucus. In bad cases, especially in entero-colitis of young children, there may be streaks of blood in the stools, but free hæmorrhage is quite unusual. The occurrence of jaundice indicates an extension of catarrh into the common bile-duct: while frequent scanty motions, containing much mucus, and accompanied with tenesmus, point to implication of the lower bowel (*dysenteric diarrhoea*). There is more or less distension of the abdomen, with rumbling sounds in the bowels. The tongue is coated, the appetite is lost,

there may be thirst, and the breath is often offensive. The urine is scanty. As a rule fever is slight or absent with adults, but in young children high fever is common.

The duration is variable. In favourable cases the attack passes off in a few days, but leaves the bowel susceptible to disturbance on slight provocation for some time. Severe cases, especially in children and aged persons, may end in fatal collapse. Lastly, it may pass into the chronic form.

A very severe form of inflammatory diarrhœa is met with in young children (*cholera infantum*) which is probably of toxic origin. It is practically confined to hot seasons, and associated with defective ventilation, together with over-heating of the body. It is probable that over-heating acts indirectly, by inducing some change in the character and functional behaviour of the bowel, which is favourable to the growth of saprophytic germs already present, but which under other circumstances would not have multiplied. A typical case is characterised by sudden onset, rapid rise of temperature to a high grade, a consuming thirst, with rapid and irregular breathing. A few hours later incessant vomiting and diarrhœa set in. From the first, the stools are frequent and liquid, and, before long, become like rice-water—odourless and colourless. The appearance of the child alters rapidly. The body shrinks, the surface becomes cold and clammy, the eyes are sunken, the fontanelles depressed, the expression set and anxious, restlessness is replaced by extreme prostration merging into coma, the voice gets weak, while fever and thirst continue. In fatal cases the child passes into a state of collapse; while in favourable cases the vomiting ceases, the fever subsides, leaving the bowel in a catarrhal condition from which convalescence is always slow; and without great care relapse may occur.

Chronic enteritis may be a sequel of the acute form, but more often it occurs independently and develops gradually.

It may be general, or more or less confined to certain parts of the bowel, especially the colon. It is a common result of improper feeding, chronic constipation, misuse of aperients, chronic congestion, or organic disease. It is most frequent at the two extremes of life. It is characterised by abdominal discomfort, fullness, and distension, associated with flatulence and colicky pains, occurring at irregular times, independently of food, and often at night or on waking in the morning. The bowels may be regular ; but, when the whole intestine is involved, diarrhœa is the rule ; while if the colon is chiefly affected, the bowels are usually costive, or attacks of diarrhœa may alternate with constipation. The nature of the stools varies. In many cases they are large in quantity, and contain a good deal of fat and undigested food. In colon cases (*simple colitis*) much mucus may be passed ; and, if erosions or ulceration be present, it may be mixed with blood.

COLITIS.

Apart from the *simple* form of colitis referred to in the preceding section, there are two special independent affections of the colon, which (for want of a better term) must also be regarded as forms of colitis. They are *ulcerative colitis*, and *mucous* or *membranous colitis*.

Ulcerative colitis is a condition closely resembling dysentery, but without any known relation to climate, season, or water-supply. Its cause is obscure. It is characterised anatomically by large irregular ulcers involving a variable length of the colon. Persistent colicky pain, often severe, is a leading feature, associated with the passage of offensive and frequent stools of a lenteric type. Blood and mucus are often present, but are not prominent features, and may be absent. It is accompanied with irregular fever, anæmia, wasting, and progressive weakness. There may be some improvement for a time under treatment, but recovery

seldom occurs, and the disease usually proves fatal within a year from the onset.

Mucous or *membranous colitis* is not an inflammatory affection, but is thought to be a secretory neurosis of the colon. It is very chronic in its course, very troublesome to treat, and is most common in neurasthenic middle-aged women. It is characterised by chronic constipation of the spastic type, and the expulsion with the stools from time to time—sometimes at long intervals—of jelly-like mucus, membranous flakes, or tubular casts of the bowel. The latter consists of structureless albuminous matter, containing many bacteria, but no leucocytes. There is always more or less abdominal discomfort, and in many cases actual pain, which may be severe and paroxysmal. Irregular pains in various parts of the body are also common, especially after exertion of any kind. The general symptoms are very indefinite and of a neurasthenic order. It is difficult to say when recovery has taken place, because, after long intervals of freedom, relapse is so common.

TREATMENT OF INTESTINAL INFLAMMATION.

In acute cases, rest in bed is desirable as a general rule. At the outset for a while nothing should be given by the mouth except sips of water-gruel or barley-water, following on with milk diluted with barley- or soda-water. If milk cannot be tolerated, whey, weak broths fortified with plasmon, or pounded meat may be tried. As already pointed out, diarrhoea from irritating matter, or from mechanical congestion, should not be checked at first, as it usually ceases spontaneously on removal of the cause. If persistent, one should try to stop it, and for this purpose aromatic chalk or full doses of bismuth—with or without opium—are best. With evidence of abnormal fermentation, salol, iodoform, or β -naphthol may be useful. When there is tenesmus, accompanied with mucus in the stools,

indicating that the lower colon is involved, great benefit is derived from occasional irrigation of the colon by full injections of warm normal saline solution. In intense choleraic cases, a hypodermic injection of morphia (gr. $\frac{1}{4}$), with atropia (gr. $\frac{1}{100}$) should be given at once, and repeated as required; and alcoholic stimulants should be given internally.

In infantile enteritis, the regulation of the diet is of the very first importance. Milk, freely, diluted, with a little lime water, will sometimes suit; but it often happens that milk cannot be tolerated in any form, and in such cases thin barley- or rice-water, or dilute solution of Mellin's food, with the addition of raw meat juice, solution of white of egg, or chicken or veal broth may be tried with advantage. With incessant vomiting it is useless to attempt feeding by the mouth; and under these circumstances rectal injections of warm normal saline solution should be resorted to. A hypodermic injection of morphia (say, gr. $\frac{1}{50}$) will sometimes check both the vomiting and diarrhœa—and thus enable other remedies to be used. In ordinary cases, nothing is better medicinally than full doses of bismuth sub-nitrate, suspended in tragacanth mucilage and aromatic water. In order to relieve pain and excessive irritability of the bowel, opium is desirable. It should never be given as a matter of routine, in no larger dose than absolutely necessary, and should be stopped as soon as its object has been fulfilled.

In *chronic* cases, much depends on careful dieting, abundant rest (especially after food), and protection from cold. In diarrhœal cases boiled milk and cereals answer best for food. Meats are not well borne. As to drugs, probably salicylate of bismuth (with the addition of an opiate, if really necessary) is one of the most useful. In cases with constipation, simple enemas are indicated, with fractional doses of calomel by the mouth.

Where the trouble is chiefly located in the colon, most reliance should be placed upon local treatment by rectal injection of warm water, to which boric acid, salicylic acid (gr. x. to the pint), or ichthyol (3 oz. to the pint) may be added. If ulceration is suspected, brilliant results may sometimes be obtained by nitrate of silver injections (gr. x.-xx. to the pint) after the bowel has been well cleared out by a simple enema.

The treatment of mucous colitis is the same as that for spastic constipation (page 384).

During convalescence great benefit will generally be derived from change to a fresh bracing air, and by tonic medicines. Further details cannot be given here, and the reader is referred to special works on the subject.

APPENDICITIS.

Inflammation of the vermiform appendix is the result of bacterial infection usually acting in co-operation with local irritation of the cæcum from fæcal accumulation, the presence of undigested food or a foreign body, or the presence of foreign matter in the appendix itself. Exertion and effort also rank as important predisposing causes in certain cases. The mucosa becomes thickened, causing obstruction of the lumen, and behind the obstruction the tube may become greatly distended by accumulated secretion and inflammatory products. Its peritoneal covering then becomes inflamed, and contracts adhesions with the surrounding parts. The further course varies. In some cases the obstruction subsides, the contents of the appendix escape into the cæcum, and recovery takes place (*simple appendicitis*), or recovery may be incomplete, and the attacks recur from time to time (*recurrent form*). In other cases, ulceration or gangrene takes place, followed by perforation of the appendix (*perforative appendicitis*).

The contents may escape and set up general peritonitis, but more often, owing to pre-existing adhesions, the effect is restricted, and localised intra-peritoneal suppuration takes place. Localised suppuration may also occur independently of perforation, from direct infection. The abscess thus formed generally occupies the right iliac fossa ; and, if left alone, bursts either externally, or internally in the peritoneal cavity, or into some other part of the bowel, or into the sub-peritoneal tissue, burrowing in various directions. The disease may occur at any age, but is most common between 10 and 30 years. Males are more often attacked than females.

SYMPTOMS.—In the mildest cases the symptoms may be limited to the sudden occurrence of pain and tenderness in the right iliac fossa, lasting for a few hours or longer, and accompanied with slight fever, perhaps occasional vomiting and then passing off. Ordinary *simple appendicitis* sets in with severe aching or tearing pain in the belly, which is at first fairly general, but gradually becomes more and more definitely referred to the right iliac region. The pain may be preceded by some flatulence and constipation, or it may begin abruptly. It is attended with faintness, clammy perspiration, and chilly extremities, or (in severe cases) with actual collapse. Nausea and vomiting are of common occurrence, but vary much in degree and duration. Fever is constantly present, soon reaching a maximum and then subsiding gradually. It usually lasts about three or four days ; and, if prolonged beyond this, is suggestive of suppuration. The abdomen is somewhat tumid and rigid, with special resistance in the iliac fossa ; in which locality, in a large majority of cases, a more or less definite tumour may be felt before long with dullness (often only relative) on percussion over a corresponding area. There is also marked tenderness over the iliac region, especially at a point about $1\frac{1}{2}$ inches from

the ant. sup. iliac spine, along a line drawn thence to the navel (MacBurney). It should be noted that this tender point does not vary according to the position of the appendix, but is a reflected superficial tenderness with a constant and definite relation to the nervous connections of the part. In addition to the above signs and symptoms there is more or less prostration, with the constitutional disturbance common to the febrile state. The bowels are usually constipated; and, in some cases, there may be irritability of the bladder. The urine is scanty, and may contain albumen. In the course of about a week, in a large proportion of cases, the symptoms abate and gradually pass off; but deep-seated induration may be felt on palpation of the iliac fossa for some time longer. As already pointed out, the attacks are liable to recur, even after a long interval.

Perforative appendicitis supervenes, as a rule, upon the previous variety, at periods varying from a few hours to two or three days. If the perforation is sudden and into the general cavity of the peritoneum, the symptoms and signs of acute peritonitis rapidly develop, and collapse follows. More often it gives rise to local intra-peritoneal suppuration. The recognition of suppuration, whether of perforative origin or not, is often extremely difficult. The chief evidence of its having occurred is as follows:—steady persistence or increase of the signs and symptoms, persistence of fever or increase of temperature after it has begun to fall, steady maintenance of a frequent pulse, marked prostration with the appearance of severe illness, and a progressive leucocytosis. Irritability of the bladder and diarrhœa are also more common than in the simple form. Digital examination of the rectum may reveal a fluctuating tumour in some cases. But after all there are many fallacies; and the greatest caution is needed to avoid mistakes. Without operative interference the abscess

bursts either externally or internally, or burrows in various directions, giving rise to complex and serious conditions.

Among the affections with which appendicitis may be confounded are—renal and biliary colic, intestinal obstruction, tuberculous peritonitis, perforating ulcer of the stomach or duodenum, or typhoid fever.

TREATMENT.—At the outset of an attack, the patient should promptly take to his bed, and remain there. For 24 hours it is well to limit the diet to barley-water, white-of-egg water, and broth or beef-tea; but afterwards peptonised milk, or equal parts of hot milk and Vichy water may be allowed. No solid food should be permitted till the approach of convalescence, and then only with caution. Hot fomentations should be applied to the abdomen; and, unless specially contra-indicated, a moderate opiate or hypodermic injection of morphia may be given to relieve pain and restlessness. As soon as this is achieved, an enema should be given; but it is now held that aperients of all kinds by the mouth should be avoided. If any medicine is employed it should be limited to a simple diaphoretic saline. With much prostration a little champagne may be sipped, but stimulants are better avoided as a rule. If the condition be not thus obviously relieved in the course of a day or two, and the evidence points to a progressive lesion, operative interference is advised, whether there be definite indications of suppuration or not. But, on the other hand, it is a mistake to suppose that surgical treatment is required in all cases of appendicitis, as a matter of course.

During convalescence and afterwards the diet and action of the bowels require close attention; and the patient should avoid violent exercise and exposure to cold and damp.

INTESTINAL OBSTRUCTION.

Intestinal obstruction arises in many different ways. The bowel itself may be blocked by impacted fæces, gall-stones, or foreign bodies ; or it may be constricted by disease of its coats, such as cancer or cicatricial contraction ; or its anatomical relations may be disturbed by intussusception, volvulus, or displacements. In another group of cases obstruction may depend upon causes outside the bowel, such as hernial strangulation, compression by tumours, adhesions, or fibrous bands.

ANATOMICAL CHARACTERS.—Any acute strangulation or obstruction is rapidly followed by extreme distension above the seat of trouble, and is attended with active congestion of the part, passing on to inflammation and gangrene, if not relieved. In chronic obstruction, the bowel above the lesion is dilated and hypertrophied, with evidence of chronic catarrh, and the peripheral portion becomes empty and contracted.

Intussusception arises from the slipping of one part of the bowel into that immediately below it. The intussusception is thus made up of three tubes, the outer and middle of which are in contact by their mucous surfaces, and connected with each other at their upper ends ; while the inner and middle tubes (*intussusceptum*) are in contact by their serous surfaces and connected at their lower ends. A fold of mesentery is imprisoned between the inner and middle tubes, which drags the *intussusceptum* to one side, so that the orifice of the latter is turned towards the outer tube.

Two varieties must be distinguished. One variety is only met with *post mortem*, and is usually multiple. It is associated with the death-struggle, and causes no symptoms during life. The other is the so-called *in-*

flammatory intussusception. The most common form is that in which the ileo-cæcal orifice forms the lowest part of the *intussusceptum*, and as it passes into the colon drags down more and more of the ileum to an extent varying from a few inches to several feet, so that it may even protrude at the anus.

The following effects will be observed, according to the severity of the case and the rapidity of its development :—*Compression* at the neck of invagination by the outer tube ; intense mechanical *congestion* followed by cedema or extravasations which still further increase the obstruction ; violent local *inflammation*, sometimes followed by *gangrene* of the *intussusceptum*.

SYMPTOMS.—The general indications of intestinal obstruction are *colicky pains* in the abdomen, coinciding with increased and sometimes visible peristalsis ; absolute *constipation*, abdominal distension ; *vomiting* which soon becomes bilious, and afterwards often fæcal in odour and colour ; and *hiccough*, followed by signs of peritonitis with frequent thready pulse, and ending in fatal collapse. When the obstruction is high up, the excretion of urine is greatly diminished, and may be temporarily suspended. Indicanuria, arising from absorption of the products of proteid decomposition, may be observed ; and slight albuminuria is sometimes found.

Symptoms of obstruction may develop suddenly without warning (*acute obstruction*) ; or gradually, with evidence of pre-existing local disease (*chronic obstruction*). Moreover, they vary considerably in detail according to the seat of the obstruction and the cause to which it is due.

DIAGNOSIS.—1. *Seat of Obstruction.*—The discovery of an abdominal, pelvic, or hernial tumour, or of a rectal constriction within reach of the finger, may at once settle the question, but frequently no information of this kind is available.

Upper part of the small intestine.—Obstructive symptoms

are marked and appear early ; tympanitis is often absent, and if present it is limited to the front and upper part of the abdomen and always relieved by vomiting ; and the vomit is bilious but never fæcal. *Lower part of small intestine.*—Obstructive symptoms appear early, and the course is rapid ; tympanitis is marked, especially in front, the flanks being relatively flat ; vomit soon becomes fæcal ; active peristaltic movements can often be seen and felt (especially in chronic cases) ; the excretion of urine is greatly diminished ; marked indicanuria may be present as early as the second day ; and, in the large majority of cases, shock is well marked from the outset. The bowels may be relieved once or twice, either naturally or by enema, before absolute constipation is established. *Colon.*—Obstructive symptoms are less marked and develop more slowly, and stercoraceous vomiting is not developed until late, or may fail altogether in obstruction of the sigmoid flexure or rectum. Distension is at first confined to the flanks, giving the abdomen a flattened contour ; but later on, the distension is general. The urine is not diminished, and indicanuria when it occurs is late in appearance. The symptoms of collapse are less striking and later in occurrence. Further information may possibly be gained by digital examination of the rectum, and by use of bougies or injections.

2. *The cause of obstruction* is often difficult to determine, and sometimes impossible. If the onset is sudden, we should think of hernia, strangulation, intussusception, or gall-stones ; or if gradual, of cancer, stricture, or fæcal impaction. A history of chronic constipation, alternating with diarrhœa, is suggestive of fæcal impaction, cancer of the rectum or sigmoid, or volvulus. A previous history of intestinal ulceration would suggest cicatricial stricture. Then as to age. In children under ten years of age intussusception is almost the only cause of acute

obstruction, while cancerous growths only occur in the latter half of life. Careful examination of the rectum and the usual seats of hernia is essential.

Impaction by faecal masses is most frequent in elderly people, and is generally preceded by habitual constipation. It is accompanied with abdominal distension and vomiting, and a soft, painless faecal tumour may often be made out. Occasionally it becomes complicated with twisting or displacement of the loaded gut, which falls over by its own weight, and in this way the signs of acute strangulation may supervene. Impacted gall-stone may sometimes be diagnosed when elderly persons exhibit signs of obstruction with previous history of hepatic colic.

Constrictions and *compressions* are gradual in their progress, and are often preceded by gradually increasing constipation with (when affecting the lower bowel) small cylindrical stools. Aid may be derived from the previous history.

Strangulations are acute in their onset, and rapidly lead to complete obstruction followed by inflammation, gangrene, or perforation. They are practically confined to the small intestine, and are more common over thirty years of age.

Intussusception is most common in children. The diagnosis rests upon the sudden onset, with violent intermittent colic, bloody mucous stools with tenesmus, in association with a movable abdominal tumour, and (in some cases) the detection of the intussusceptum by digital examination of the rectum.

As regards the general diagnosis of acute obstruction, acute peritonitis—however it may originate—is most likely to cause confusion; indeed, a diagnosis can never be made without a very careful study of the signs, symptoms, and history of the case; and even then mistakes are common enough. Lastly, it may be pointed out that in acute

obstruction the diagnosis is attended with serious practical difficulties, and is often impossible later than the second day from the onset.

TREATMENT.—Acute obstruction, such as that depending upon strangulation, calls for prompt surgical treatment. In less acute cases, and where obstruction is incomplete, enemata should be freely given to unload the bowel below the point of obstruction, but purgatives must be avoided. If no food can be retained when taken by the mouth, nutrient enemata are required. Unless contra-indicated by extreme prostration, lavage of the stomach is sometimes of great service, especially when resorted to early. For the relief of pain and vomiting, subcutaneous injections of morphia may be necessary; but no larger dose should be given and not more often than just sufficient to complete the desired end, for it masks the symptoms, makes diagnosis more difficult, and is apt to produce a false sense of security at a time of imminent danger. Atropia may be freely combined with the morphia. It gives relief to pain and spasm, even if used alone. In the early stages of intussusception it may be of service to put the patient under an anæsthetic, and, with the hips raised, to administer a copious injection of warm, normal saline fluid under considerable pressure. Thirst and vomiting may be relieved by sucking ice, and hot fomentations to the abdomen are very soothing. If the above measures are not speedily followed by relief, operative interference should not be delayed. Probably many a case is lost for want of the moral courage to resort to an operation until the favourable opportunity has passed by.

CANCER OF THE INTESTINES.

Cancer of the intestines is almost always primary, and is most common by far in the colon, especially in the rectum or sigmoid flexure, but seldom in persons under forty years

of age. Various types of cancer are met with, but most frequently it takes the form of fungous out-growths from the mucous surface, displaying abundant tubular collections of cylindrical epithelium on microscopic examination. As a result of infiltration and contraction of the bowel, intestinal stricture is of common occurrence. Secondary deposits are apt to occur in the liver and other parts.

The *symptoms* of intestinal cancer may be so vague and insignificant as not to excite attention, until perhaps obstruction or hæmorrhage supervenes. But usually we meet with abdominal pain and sometimes local tenderness, together with obstinate progressive constipation, or irregular action of the bowels, with offensive stools, often containing mucus or blood. When the disease is low down there may be abnormal formation of the stools in small lumps, ribbons, or pencil-like cylinders. Examination of the abdomen may reveal, in addition to distension, a hard, irregular tumour in some part, but care should be taken not to mistake a fæcal accumulation for a new growth. As occasional symptoms, mention may be made of hæmorrhage from the bowel, intolerable irritation of the anus, and pain or swelling of one or both legs. Sooner or later the general health suffers, with progressive weakness and emaciation. Intestinal obstruction is a common ultimate result, and is typically of a chronic type; yet when it has reached a certain point, a sudden impaction of a fæcal mass may bring on an acute obstruction. The condition is so often overlooked that a digital examination of the rectum should always be made on any complaint of persistent abdominal or rectal discomfort in elderly persons. The duration of the disease is variable, but it seldom exceeds a year and a half, when death occurs from exhaustion, or from obstruction, or from some complication.

The *treatment* is purely palliative and symptomatic. An artificial opening into the colon prolongs life in certain cases.

INTESTINAL TUBERCULOSIS.

Intestinal tuberculosis in adults is usually secondary to pulmonary phthisis, and is probably due to inoculation from swallowed sputa. In children, however, it is often associated with caseous mesenteric glands and tuberculous peritonitis. The solitary and agminated follicles of the bowels are first attacked. They become enlarged and afterwards caseate and break down, forming an ulcer with indurated floor and margins. The ulceration extends much more rapidly in a transverse than in a longitudinal direction, so that it sometimes forms a complete ring around the inside of the gut. Tubercles are found in the tissue forming the edge and floor of the ulcer, which in their turn degenerate and break down, and constitute the mode by which the ulceration extends. Inflammatory adhesions form between adjacent coils of intestine at the seat of the ulceration, and sometimes a perforative communication is set up between them. Or, again perforation may lead to a local intra-peritoneal abscess. The ulcers have little tendency to heal, though occasionally imperfect cicatrization occurs, and causes some intestinal constriction.

More or less abdominal pain and tenderness, with obstinate diarrhœa, and bloody stools observed in a patient suffering from phthisis should at once suggest tuberculous implication of the intestine. But in some cases there are no characteristic intestinal symptoms. In a child over two years of age, persistent irregular fever, a distended abdomen, chronic intractable diarrhœa, marked wasting and pallor, especially if accompanied with irregular resistance over the abdomen and tenderness on pressure, would point to tuberculous disease of the bowel.

The course of the affection is progressively fatal, and treatment can only be palliative.

INTESTINAL WORMS

The worms which are found in the intestinal tract may be divided into two main groups:—1. The *Tæniada* or *Cestoda* or *Tape-worms*, including (a) *Tænia solium*, (b) *T. medio canellata*, and (c) *Bothriocephalus latus*. 2. The *Nematoda* or *Round worms* including (a) *Ascaris lumbricoides* (round worm), (b) *Oxyuris vermicularis* (thread-worm), and (c) *Trichina spiralis*.

1. TÆNIADA.—*General Remarks*.—All members of this group have two phases of existence. Segments of the fully developed worm containing ripe ova become detached, and on being discharged from the body are scattered about. Sooner or later they find their way into the intestine of some appropriate animal. The embryo contained within each ovum then bursts its shell, passes through the intestinal wall, and works its way into the tissues. On reaching a suitable spot it further develops and presents a *head* resembling that of a perfect worm, a short *neck*, with a vesicular or bladder-like appendage (*cyst*) filled with clear fluid. In this stage it is called a *cysticercus*. It possesses no sexual organs, and is only capable of further development on its removal into the human intestine. This takes place when the tissue containing the cysticercus is eaten with food. Under these circumstances it fixes itself to the intestinal mucous membrane, drops its vesicular appendage, and develops from its hinder end the series of segments which characterise the adult worm. Thus *two distinct stages* are needed to complete the cycle of existence of these worms.

(a) *Tænia solium*, or the common tape-worm, is of an elongated, flat shape, from five to ten feet in length, and dwells in the small intestine. Its head (*scolex*) is about the size of a pin's head and is furnished with a circle of

hooks surrounded by four suckers, by which it attaches itself to the intestine. From the head backwards it is made up of a series of soft white segments (*proglottides*), at first very small (*the neck*), but gradually enlarging as they become more mature. The segments of the neck are much broader than long, and nearly homogeneous; but proceeding backwards they gradually become longer than they are broad ($\frac{1}{2}$ in. $\times$ $\frac{1}{4}$ in.) ; and about one foot from the head, male and female sexual organs are visible, which open into a common aperture, and contain ova. Usually only one worm is present in the intestine at the same time.

Its *cysticercus* (*cyst. cellulosus*) gives the well-known appearance of "measly" pork, from eating which when badly cooked, tape-worms exclusively arise. The head exactly resembles that of *tænia solium*, and is joined to a roundish vesicle about the size of a pea by a short wrinkled neck.

(b) *Tænia mediocanellata* very closely resembles the former species, but it is longer, its head is three times as large, and it is not provided with hooklets. The *cysticercus* of this worm especially affects the ox, and is apt to be introduced into the human body by eating badly cooked beef. It is of very common occurrence.

(c) *Bothriocephalus latus*, in Europe, is peculiar to Switzerland and the Baltic shores; and is the largest of all tape-worms. The head is club-shaped, with a longitudinal slit by which it attaches itself, and has no suckers or hooks. Its *cysticercus* is not definitely known, but is supposed to inhabit fish.

SYMPTOMS.—In many cases there are no symptoms whatever. In others, however, there are indications of more or less nervous and digestive derangement. Thus pains in the bowels, irregular appetite, thirst, headache, troubled sleep, vertigo, mental depression or irritability, and itching of the anus or nose are common symptoms. More rarely,

the nervous disturbance assumes a severe type. The appearance of the ripe segments in the stools is characteristic.

TREATMENT.—The patient should be kept on a very spare diet for twenty-four hours, an aperient should be administered in the evening, and as soon as the bowels have acted next morning at least $\mathfrak{zj}$. of the extract of male fern should be taken on a fasting stomach, either in capsules or in some simple emulsion flavoured with peppermint water. No absolute cure is effected unless the *head* is expelled. Unless the head is found in the stools, it is not safe to assume that a cure has been effected, until the lapse of three months without signs of return. The bark of the pomegranate root, kousso, or oil of turpentine have also been recommended, but are inferior to male fern.

2. NEMATODA.—*General Remarks.*—The members of this group are solid and rounded in shape, and possess a distinct alimentary canal provided with mouth and anus. The sexes are always separate. It appears now to be established that the life history of these worms does not involve an intermediate host.

(a) *Ascaris lumbricoides*, or common round worm, closely resembles in its form the common earth worm. The female is nearly twice as large as the male, measuring eight to twelve inches in length. It is cylindrical, pointed at each end, and of a white colour. The ova, of which thousands are discharged daily, are oval, about $\frac{1}{400}$ in. diameter, with a thick irregular shell. More than five or six worms are rarely present at the same time. Their special habitat is the small intestine, but they are peculiarly apt to wander in every conceivable direction.

SYMPTOMS.—These may be altogether wanting, or when present may be in no way characteristic, consisting chiefly of slight fever, disturbed sleep, thirst, and depraved appetite, with itching of the nose and anus. Occasionally a mass of them may give rise to temporary intestinal obstruc-

tion ; or they may be vomited. Ova may be readily detected by the microscope in the stools.

TREATMENT.—The best remedy is *santonin* (gr. j.-iij. for a child, or twice this quantity for an adult), which should be given for three successive evenings. Castor oil, or a bitter aperient such as decoction of aloes, should be given from time to time.

(b) *Oxyuris vermicularis*, or common thread-worm, is small, white, and thread-like. The female is twice the length of the male and measures from $\frac{1}{3}$ to $\frac{1}{2}$ inch. They dwell in the cæcum, colon, and rectum. They are the most common of all worms, are chiefly met with in badly fed children, and always exist in great numbers. They are apt to migrate through the anus, and may find their way into the vagina, setting up leucorrhœa or eczema.

SYMPTOMS.—The most important sign is a troublesome itching about the anus, worse at night, and which is sometimes almost intolerable. There may be also itching at the nose, irritability of the bladder, digestive disturbance, dry barking cough, and indifferent general health. The worms and their ova are readily detected in the fæces.

TREATMENT.—The expulsion of the worms by the regular employment of aperients, is a primary indication. For this purpose, sulphur, made into an electuary with treacle, is best for children, and the compound decoction of aloes is best for adults. Meanwhile, a full enema of salt-solution should be given once or twice a week. Careful regulation of the diet is also of great importance, for in obstinate cases there is always intestinal catarrh, without the cure of which, riddance from thread-worms is impossible. All easily fermentable articles of food, such as sugar, starches, pastry, and fruits, should be avoided, or taken in strict moderation. Milk, meat, and fish should form the staple diet, and nothing should be taken between meals. In this connection also, medicinal tonics such as quinine, iron, and

nux vomica are of service. If the rectum be prolapsed, it should be bathed with cold water, smeared over with glycerine of tannin, and replaced. Daily enemata of cold water will help to give tone to the bowel.

(c) TRICHINA SPIRALIS.—*Trichinosis*.—This affection is due to eating trichinous pork. The larvæ of the *Trichina* occur in muscular tissue, coiled up within tiny cysts, the walls of which (in man) are thick, laminated, and more or less calcareous; but in pigs, calcification occurs much less readily, and thus the larval cysts are less obvious to the eye. On such pork being eaten, the cysts are dissolved by the gastric juice, the embryos are liberated, and in a few days develop into mature worms—male and female. The females, which are about $\frac{1}{8}$ in. in length, burrow into the wall of the intestine, discharging thousands of ova, which are carried in the circulation to the muscles, where they finally lodge, and gradually form the larval cysts above described.

SYMPTOMS.—There may be some prodromal gastro-intestinal disturbance shortly after injection, but characteristic symptoms are usually delayed until some time in the second week. They consist of acute gastro-intestinal disturbance not unlike typhoid fever, with coated tongue, diarrhœa, pain in the bowels, and fever. In the course of a few days, signs of general muscular inflammation supervene, such as pain, tenderness, swelling, and stiffness of the voluntary muscles. Dropsy soon develops, beginning in the face and particularly in the eyelids, then extending to the limbs, and subsequently becoming general.

The duration and severity of trichinosis vary greatly. In mild cases, recovery may take place in the course of a month, in others after three or four months.

In other cases again, the mortality is high, and death occurs from peritonitis, pneumonia, or pleurisy at any time between one and six weeks.

If the introduction of the parasites into the body be discovered quite early, prompt administration of a brisk aperient is likely to expel them before they have entered the tissues. Otherwise, the indications for treatment are purely symptomatic, as no specific remedy is known.

SUB-SECTION V.

DISEASES OF THE PERITONEUM.

ACUTE DIFFUSE PERITONITIS

Acute diffuse peritonitis occasionally results from general disease such as septicæmia, influenza, Bright's disease, or gout; but far more commonly it arises from local causes, such as from perforation of the appendix, stomach, or bowel, or from infective inflammation of the Fallopian tubes or ovaries. It is also not an infrequent sequel of surgical operations upon the abdomen or its contents. It is almost invariably of microbic origin; and the organisms which are chiefly responsible are streptococci, staphylococci, colon bacilli, pneumococci, and various putrefactive bacteria. Here and there, however, it may depend upon a toxic cause, as in Bright's disease.

MORBID ANATOMY.—There is congestion of the peritoneal membrane, followed by exudation of lymph with or without fluid. The lymph is yellowish, and forms a layer of variable thickness which leads to adhesion between its opposed surfaces. A fluid effusion follows, but the patient seldom survives long enough for it to become abundant. It is more or less turbid with flakes of lymph in suspension, or actually purulent.

SYMPTOMS.—The onset of acute diffuse peritonitis is sometimes abrupt, and in the perforative variety is characterised by sudden collapse; but not infrequently the onset

is insidious. In any case severe abdominal pain and tenderness quickly supervene; and the patient lies on his back with his knees drawn up, dreading the least pressure or movement. The pain is general at first, or most marked about the umbilicus, but later on it often becomes more localised to some definite part of the abdomen. Nausea and vomiting are often distressing. The expression betrays extreme anxiety, the features are pinched and drawn, and the general condition is indicative of more or less collapse. The abdomen is at first rigidly contracted; but later, it usually becomes distended and tympanitic, and the liver dullness may be obliterated. There may be more or less effusion of fluid into the peritoneal cavity. The temperature is sometimes high, especially at the onset, but more often it is moderate, and in cases attended with much collapse it may be wanting altogether. The tongue is at first coated and moist, but afterwards becomes dry; there is great thirst, and the appetite is lost. The bowels are usually constipated, but sometimes (especially in puerperal peritonitis) they are relaxed. The pulse is rapid, weak, and small; the respiration is hurried and shallow; the urine is scanty, and the bladder is sometimes very irritable. As a rule there is but little nervous disturbance, and the mind remains clear.

In exceptional cases the above symptoms may subside, and recovery follow, but far more often it is fatal—signs of rapidly increasing collapse supervene, though the pain and tympanitis may diminish or disappear; the skin becomes cold and clammy; the pulse becomes more frequent, weaker, and often irregular; and now nervous symptoms set in, though the mind may be clear to the end.

The diagnosis of acute diffuse peritonitis in typical cases is usually not difficult. Acute obstruction of the

bowel, however, may at times give rise to very great difficulty in diagnosis; and, among the rarer sources of confusion, acute pancreatitis should be mentioned.

TREATMENT.—As a result of very considerable advance in the methods of abdominal surgery, the surgical treatment of peritonitis has acquired great importance. Not only in those cases in which there is some obvious surgical condition such as strangulated hernia, but in all cases of perforative and purulent peritonitis, operative interference is now uniformly advised, provided it can be carried out early enough.

Otherwise we have to rely upon the following therapeutic measures. The patient should be in bed in the position most comfortable to him, and protected from the pressure of the clothes by means of a bed-cradle. As little should be given by the mouth as possible—nothing more as a rule than an occasional spoonful of some simple watery drink just to mitigate intolerable thirst. Something may also be done in this direction by the rectal injection of half a pint of warm water from time to time, and by keeping the mouth scrupulously clean and moist. If vomiting be slight, a little light liquid nourishment may be attempted by the mouth, but in the main we must rely upon rectal feeding. Hypodermic injection of morphia may be given, but only in just sufficient quantity to relieve pain and restlessness, so long as there is any chance of saving the patient. When they can be borne, hot and moist fomentations, poultices, sinapisms, or stupes, may be employed; while others again prefer evaporating lotions, cold compresses, and ice-bags. Under no circumstances should active purgatives be given; but in the *early* stages of peritonitis, especially after abdominal operations, saline aperients are sometimes beneficial.

CHRONIC PERITONITIS.

As a *local* affection, chronic peritonitis is of common occurrence, giving rise either to fibrous adhesion of the spleen or liver to adjacent parts, with great thickening of their capsules, or to matting together of certain parts of the bowel, as sometimes happens in chronic duodenal ulcer. More often it is only discovered *post mortem*; but it is sometimes attended with troublesome, irregular abdominal pains; and, in rare cases, fibrous bands may be formed, in which a loop of bowel may be caught.

As a *diffuse* affection, it may occur as a sequel of the acute form, but much more often in association with cancer or tubercle. An interesting variety of chronic diffuse peritonitis may be met with, accompanied by a tendency to corresponding chronic adhesive inflammation of other serous membrane, such as the pleura or pericardium (*polyserositis*). Multiple serous inflammations may be due to tuberculosis; but the affection in question has nothing to do with tubercle. It is more often associated with chronic alcoholism, interstitial nephritis, or arteriosclerosis, and appears to be due to some kind of endogenous intoxication.

The peritoneum is opaque, and often considerably thickened, with more or less adhesions. Serous effusion may be present, and owing to adhesions, it may be distributed in pockets, and prevented from shifting or change of posture. Perihepatitis and perisplenitis—either or both—are of common occurrence. The omentum is liable to be rolled up, and form a palpable tumour below the stomach. Or again, the mesentery may be so shortened that the intestines form a more or less compact mass.

TUBERCULOUS PERITONITIS.

Tubercle of the peritoneum may occur as part of a general tuberculosis, or as a local accompaniment of tuberculous ulceration of the bowel. But there are also cases in which it constitutes the main and essential condition, sometimes as a primary infection resulting from the direct passage from the bowel of tubercle bacilli taken with the food, but more often as a secondary infection from pre-existing tuberculous foci in the lungs, lymphatic glands, testes, or Fallopian tubes. In children it is sometimes associated with the presence of irregular masses formed by caseous mesenteric glands or omentum, a condition which is known as *tabes mesenterica*. Such masses are also occasionally met with apart from recognisable peritonitis.

Tuberculous peritonitis may occur at any age, but is more common in early adult life. Three groups of cases may be distinguished, according to the degree of adhesion; but be it understood that such distinction is purely artificial, and solely for descriptive purposes. In the first (*ascitic*) there is an effusion into the general peritoneal cavity, and adhesions are few or absent; in the second (*loculated*) adhesions are disposed so as to form pockets, with sero-fibrinous, purulent, or caseous contents; and in the third (*adhesive*) the peritoneal cavity is practically obliterated by general adhesion. In many cases the omentum becomes rolled up and adherent, forming a palpable tumour.

SYMPTOMS.—The symptoms vary somewhat according to type, but as a rule after a variable period of failing health, loss of flesh and strength, and constipation or irregular action of the bowels, attention is called to the abdomen by local discomfort or colicky pains, possibly some tenderness, and gradually increasing tumidity. When

fully established, there may be evidence of ascites, or of encysted collections of fluid indicated by irregular areas of impaired resonance on percussion, or of palpable tumours formed by pockets of effusion or caseous matter, coils of intestine matted together, enlarged lymphatic glands, or a rolled up omentum. In chronic uncomplicated cases there may be little or no fever, and very little local discomfort ; but, with suppurative or caseous lesions, there is generally more or less fever of a hectic type, with considerable local pain and tenderness. Though there may be temporary improvement, the large majority of cases end fatally from exhaustion or the development of tuberculous disease in the bowel, lung, or elsewhere. The duration varies greatly, but generally extends over several months.

Tuberculous peritonitis is by no means always easy to recognise. The acuter forms may be mistaken for typhoid fever ; the caseous form may closely simulate other types of intra-abdominal suppuration ; the earlier stages of many chronic cases may be very difficult to distinguish from chronic intestinal catarrh—especially in young children ; and lastly, it may sometimes closely resemble intestinal obstruction.

Medical *treatment* is exclusively symptomatic. Nevertheless tuberculous peritonitis is by no means a hopeless condition, though the mortality is high. A certain proportion, especially cases of the ascitic type, may recover spontaneously ; and surgical interference by tapping or by abdominal incision and drainage is sometimes followed by recovery, though the indications for its employment are not very precise.

SUB-PHRENIC ABSCESS.

Among the various forms of localised suppurative peritonitis, sub-phrenic or sub-diaphragmatic abscess is

the most important. The exact position varies according to its cause. It is also desirable to distinguish *simple* sub-phrenic abscess from that which contains gas as well as pus (*sub-phrenic pyo-pneumothorax*). The simple form more often results, directly or indirectly, from appendicitis. It is usually intra-peritoneal, lying over the liver to the right of the falciform ligament, and in front of the right lateral ligament of the liver, sometimes extending into the right renal pouch; but when secondary to hepatic suppuration, it is more often extra-peritoneal, lying over the uncovered part of the liver, between the layers of the coronary ligament. On the other hand, gas-containing abscess is generally caused by perforative ulcers of the stomach, and lies between the left vault of the diaphragm above, and the left lobe of the liver, stomach, and spleen below. Yet again, suppuration may be confined to the lesser sac of the peritoneum, when due to perforation of the posterior wall of the stomach or lesions of the pancreas. Sub-phrenic suppuration is very frequently associated with pleurisy on the corresponding side, attended with effusion, which may be sero-fibrinous or purulent; and sometimes there is actual perforation of the diaphragm.

The symptoms vary widely according to the cause and seat of the abscess. In the simple form the onset is insidious, and is more often overlooked. It follows an obscure course, and is very apt to be mistaken for empyema. In perforative cases, on the other hand, the onset is sudden, and attended with severe pain, tenderness, vomiting, and dyspnœa. Later on, as suppuration takes place, there are signs of blood-poisoning—irregular fever, sweating, and emaciation. If the abscess perforates the diaphragm or lung, there will probably be cough and expectoration. Sometimes there is a fairly well-defined prominence below the costal margin; but more often the physical signs

are very obscure, closely resembling those of an empyema or a pyo-pneumothorax, and the obscurity is accentuated by frequent association with pleural effusion. The history, however, and mode of onset point rather to the abdomen than the thorax, the liver is more depressed than is usual in pleural effusion; the heart (if displaced) is pushed upwards, and not to one side as in pleural effusion; and the signs of an air-containing cavity may be found well below thoracic limits, and hepatic dullness may be obliterated.

ASCITES.

Ascites strictly implies a *dropsical* effusion into the peritoneum, but the term is very generally employed to indicate fluid effusions of *any* kind into that cavity. The causes of ascites may be grouped in three classes. It may arise (1) as a part of general dropsy; (2) from portal obstruction; or (3) from peritoneal inflammation—especially of a chronic type. The amount of fluid varies from a few ounces to several quarts; it is usually clear, alkaline, of a yellow or greenish tinge, and contains a large quantity of albumen. Its specific gravity when of dropsical origin is generally below 1010; from portal obstruction it varies from 1010 to 1016; and when resulting from inflammation, the fluid is often turbid and flaky with a specific gravity above 1016.

SYMPTOMS.—On *inspection* the belly is found to be enlarged in proportion to the amount of effusion. In large effusions, the skin is smooth, tense, and may be marked with whitish striæ, or covered with a network of distended superficial veins. The navel is flattened out, obliterated, or everted. On *palpation* a peculiar thrill (fluctuation) may be felt on placing one hand flat on one side of the abdomen, while the opposite side is smartly tapped with

the other hand. To get this result it is necessary that both points should be immediately over the effused fluid. The *percussion note* is dull wherever the fluid is in contact with the abdominal walls, and the upper limit of the dull area assumes a hydrostatic level. Further, since the fluid sinks to the most dependent parts, and the intestines float on the surface, the distribution of the dull and resonant areas varies according to posture. Lying on the back, the flanks are dull and the neighbourhood of the umbilicus is resonant; by lying on one side the uppermost flank becomes resonant; and so on.

Other symptoms arise indirectly. Thus, dyspnœa and palpitation occur from pressure upon and displacement of the diaphragm and heart respectively, and functional derangements of the stomach and bowels are common events in ascites dependent upon portal obstruction. In some cases, the pressure of a large and tense effusion upon the vena cava may account for œdema of the legs and albuminuria, but it should be remembered that the same conditions may be brought about by other factors, cardiac, renal, or toxic.

When the effusion is scanty, recognition may be difficult; and when the amount of fluid and consequent abdominal distension are excessive, the shape of the abdomen is more globular, and there may be dullness all over the abdomen.

DIAGNOSIS.—Ascites may be confounded with an ovarian cyst, pregnancy, a distended bladder, a hydatid cyst, or hydronephrosis. The differential diagnosis cannot be discussed here in detail, and it must suffice to point out that the most typical features of ascites are the symmetrical distension of the abdomen, a flattened navel, dullness in both flanks with resonance in front, the shifting of the limits of dullness on alteration of posture, the hydrostatic level of the upper limit of dullness, and the following re-

sults of mensuration. In simple ascites (apart from œdema or thickening of the lower abdominal wall) the maximum circumference of the belly is *never below* the level of the umbilicus, and the distance between the pubes and umbilicus is *never greater* than that between the umbilicus and the xiphoid cartilage.

Having established the existence of ascites it yet remains to ascertain its cause. In the first place it is necessary to separate inflammatory from simple dropsical effusions. Some pyrexia, with pain and tenderness of the abdomen, are commonly present in greater or less degree in the former, but are absent in the latter. The nature of the fluid in each also differs as already shown. Non-inflammatory dropsical effusions may be either renal, cardiac or pulmonary, or hepatic. In the *first* the ascites is preceded by anasarca, which usually begins in the face, fluid is present as a rule in other serous cavities, the countenance is pale and pasty, and there are signs of renal disease. In the *second*, ascites is preceded by anasarca, which begins at the ankles and extends upwards, the countenance is dusky or livid, and there are signs of pulmonary or cardiac disease. In the *third*, anasarca is absent or follows the ascites, and is confined to the lower limbs; and there are the signs of hepatic disease. Cancer and cirrhosis are practically the chief hepatic diseases which are attended with ascites, and as to the diagnosis between them the reader is referred to the description of these diseases.

The *treatment* of ascites for the most part is that of the affection on which it depends, but special measures are often called for, which essentially consist in the employment of diuretics, diaphoretics, and hydragogue purges. In the selection of such remedies, due regard should be paid to the general condition of the patient; thus strong diuretics, copaiba resin for instance, are out of the question in renal disease. Paracentesis abdominis may be resorted to in

extreme effusions, but it should be borne in mind that the operation is only palliative as a rule, that its repeated performance is exhausting to the patient, and that it is often worse than useless in ascites dependent on cancer.

SUB-SECTION VI.

DISEASES OF THE LIVER.

GENERAL CONSIDERATIONS.

The functions of the liver are very complex; and, though much has been learnt about them, they are still very imperfectly understood. In one of its simpler aspects, the liver fulfils an excretory purpose by removing products of wear-and-tear from the blood, among which the most important is effete hæmoglobin, from which bile-pigment is derived. But the liver does much more than this. From its vascular relations with the alimentary canal, everything that is absorbed from the latter (with the exception of fat, which is conveyed by the lacteals) has first to pass through the liver, and be submitted to its influence before it can enter the general circulation. In this manner, the liver occupies a unique position as the presiding genius of nutrition. The power by which it exerts this influence is an inherent property of the living protoplasm of the hepatic cells, maintained by blood, representing some of the purest in the body, brought to them by the hepatic artery. Thus endowed, the cells react upon the portal blood by which they are bathed, taking up some of its constituents—nutrient, adventitious, or toxic, as the case may be, and submitting them to metabolic change; of the products of which, some are returned to the blood, while others are discharged by the bile-ducts. To sum up, the functions of the liver are partly assimilative (in so far as it subserves the

process of nutrition); partly protective (in so far as it modifies or destroys adventitious or toxic material); and partly excretory. Although it is convenient to think of these functions separately, it must be remembered that they are closely connected with each other, and vary more or less on parallel lines, so that no one function can be seriously disturbed without compromising the rest. A final word may be added, with regard to the part taken by bile in the process of intestinal digestion, which is limited to the emulsification of fats, and the promotion of their absorption in consequence.

CONGESTION AND INFLAMMATION OF THE LIVER.

HEPATIC CONGESTION.

ACTIVE CONGESTION of the liver always accompanies the digestive act, but whenever food is taken in excess, or is of an irritating nature, especially when connected with alcoholic abuse, it is apt to exceed its normal limits. Congestion also occurs in connection with malarial and other fevers, injury, or new growths. Active congestion merges insensibly into actual inflammation of the liver. The liver is enlarged, dark-coloured, and its vessels gorged with blood. The liver-cells are swollen and show signs of cloudy swelling or fatty change.

Symptoms.—The subjects of active hepatic congestion are irritable, depressed, and complain of headache and general malaise. Painful tension is felt in the right side, which often radiates to the right shoulder. The tongue is foul, the appetite is lost, nausea and vomiting are of common occurrence, and the bowels are constipated. The urine is high-coloured, and yields a copious deposit of urates. There is sometimes a little jaundice, due to

associated catarrh of the bile-ducts. The liver is uniformly enlarged, and tender on pressure.

MECHANICAL CONGESTION of the liver is due to impeded outflow of blood, which generally arises from cardiac valvular disease, or chronic bronchitis and emphysema. The liver is uniformly enlarged. The centre of each lobule is dark-coloured from engorgement of the radicles of the hepatic vein, while the cells at the periphery are pale and atrophied from pressure, thus yielding a mottled appearance of the cut surface, like that of nutmeg (*nutmeg liver*). In course of time the interstitial connective tissue is increased and afterwards shrinks, in consequence of which the organ becomes indurated.

The *symptoms* are largely merged with those of the condition on which it depends. In addition to hepatic enlargement and tenderness, there are disturbance of digestion, irregular behaviour of the bowels, and sometimes piles from portal obstruction. In the later stages ascites sometimes occurs, as the result of extensive hepatic induration.

In the *treatment* of hepatic congestion rest must be enjoined, the diet should be bland and unirritating, and stimulants as a rule are harmful. In the first instance, a mercurial, followed up by some saline aperient, such as the sulphates of magnesia or soda, tartrate of potash, Hunyadi János water, and the like, will often be found useful. Local pain and tenderness may be relieved by fomentations or poultices, or (in severe cases of mechanical congestion) by venæsection. In acute cases, a course of chloride of ammonium is often of great service. In mechanical congestion the general treatment is essentially that of the condition on which it depends.

ACUTE HEPATITIS.

It may be reasonably assumed that severe forms of acute

hepatic congestion merge into acute inflammation, and there is no need for description beyond what has been already said with regard to acute congestion. The most important form of hepatitis is that which is attended with suppuration. Three varieties require notice.

1. *Pylephlebitis*.—This somewhat rare affection is characterised by suppurative inflammation of the hepatic branches of the portal vein, associated with thrombosis, which depends upon septic infection, proceeding as a rule from some part of the portal area, usually the cæcum, appendix, or colon. It is attended with pyæmic symptoms, but scarcely admits of diagnosis at the bedside. It is always fatal.

2. *Pyæmic abscesses*.—These occur under the same conditions as pylephlebitis, or as part of a general pyæmia, and consist of small abscesses scattered through the liver, which is moderately enlarged, and very tender to pressure. The symptoms are practically the same as those of pylephlebitis, from which it cannot be clinically distinguished.

3. *Tropical abscess*, as the name implies, is most common in tropical climates, more often as the result of dysentery; but in many cases the cause is obscure, though it must be of an infective nature.

It is usually single and often of large size, causing an irregular painful enlargement of the liver. When superficial, it may yield fluctuation on palpation. It may burst into the peritoneal cavity, causing fatal peritonitis; but more often, as it approaches the surface of the gland, adhesive inflammation is set up, which glues the organ to adjacent parts, and the pus is discharged either externally, or into the lung, pleura, or stomach.

Tropical abscess may run an entirely latent course, but more often we meet with marked fever of a hectic type, dull aching pain referred to the hepatic region or

right shoulder, with more or less tenderness on pressure below the right costal border. Rigors, vomiting, and profuse sweating are of common occurrence. The complexion is of a dirty sallow colour, and the conjunctivæ may be yellowish, but actual jaundice is rare. The liver is enlarged, but not uniformly as a rule. Thus the right lobe is more often involved than the left, with a special tendency to enlargement upwards and to the right, yielding physical signs which may be not unlike those of an empyæmia. When it can be reached by the knife, opened, and drained, recovery may follow. If left to itself, it is most likely to perforate into the lung; and, in any case, the course is then tedious, and usually proves fatal. In doubtful cases, the liver may be aspirated without hesitation, to aid diagnosis.

CIRRHOSIS OF THE LIVER.

CIRRHOSIS, as a general term, signifies a morbid increase of interstitial connective tissue. In this sense, cirrhotic changes in the liver may arise in various ways—as the result, for instance, of chronic mechanical congestion, of local inflammatory or degenerative lesions, or of syphilis. But, apart from these, there are two morbid conditions of the liver in which more or less generalised fibrosis occurs, to which the term “cirrhosis” is applied in a more special sense. The one is fairly common, is generally due to alcoholic abuse, and is attended with signs of portal obstruction (*portal cirrhosis*). The other is less common, occurs quite independently of alcoholism, is not attended with portal obstruction, and is associated with chronic jaundice (*biliary cirrhosis*).

PORTAL CIRRHOSIS.

Alcoholic excess, especially in regard to spirits, is un-

doubtedly the commonest cause of portal cirrhosis. But it is sometimes set up by other poisons introduced from without, or toxins produced within the body. The exact mode in which alcohol or other causes act is somewhat uncertain, but it may be partly direct and partly indirect. At any rate, it is quite clear that the disease is of toxic origin, and that the *materies morbi* reaches the liver by the portal vein. Susceptibility to portal cirrhosis probably varies considerably in different persons. It is essentially a disease of adult life, and is far more common in men than in women.

Morbid Anatomy.—The size of the liver varies greatly. Sometimes it is small, but more often it is enlarged. The cause of these variations has been much discussed, and probably it is not always the same. Thus enlargement may be due to vascular engorgement, to fatty infiltration, or to compensatory hyperplasia of liver cells. But whether the liver be large or small, the disease is essentially the same. The surface is usually covered with projections which are sometimes small and uniform, and in other cases large, irregular, and separated by deep cicatricial furrows, leading to deformity of the organ. More often, however, especially in large livers, the surface is relatively smooth, or indistinctly granular. The consistence is hard. On section, the surface is usually of a pale yellow colour, and is mapped out in multi-lobular areas of variable size by a fibrous mesh-work.

The hepatic artery and its branches, together with the larger branches of the portal vein, are distended; but many of the smallest branches of the latter are compressed and thrombosed, and some of the radicles of the hepatic vein may be obliterated.

As a result, the portal circulation is obstructed, causing congestion of the parts which are drained by the portal vein. Under these circumstances the portal blood over-

flows into the systemic veins and causes dilatations at the following point of anastomosis:—at the junction of the œsophageal with the gastric veins, of the hæmorrhoidal with the mesenteric veins, of the mammary and epigastric veins with those in the round and falciform ligaments of the liver, and of the retro-peritoneal with the mesenteric veins.

On microscopical examination it will be seen that the normal arrangement of the so-called lobules is seriously disturbed. They are much more unequal in size and shape; the cells are no longer grouped in uniform radial fashion around a central vein; the latter is often eccentric, and some groups of cells appear to have no central vein at all. These results are due to atrophy or actual necrosis of old hepatic cells, accompanied with a very considerable formation of new ones, which takes place chiefly at the periphery of the lobules. Added to which, in common with all hepatic lesions attended with destruction of liver-cells, a large number of newly formed biliary canaliculi are found. The increased formation of connective tissue mainly encloses and partly invades the lobules, for the most part in groups, hence the term multi-lobular is sometimes applied to this form of cirrhosis.

The spleen is commonly enlarged, partly as the result of portal obstruction, and partly of hyperplasia occurring under toxæmic influence. Ascites, which is always present, sooner or later, is due to portal obstruction, and partly also to inflammatory and toxæmic factors. Arteriosclerosis, renal disease, tuberculosis of lungs or peritoneum, or alcoholic neuritis may be associated with cirrhosis.

Symptoms.—The onset is usually insidious, and little is noted beyond dyspeptic trouble, and possibly some discomfort and tenderness about the liver. Among the earlier symptoms, hæmatemesis is the most important, though it only occurs in about a quarter of all cases. It

results from rupture of varicose œsophageal veins near the cardiac orifice of the stomach as a rule. It is often profuse, but seldom directly fatal. It may recur, but more often it is limited to a single attack. Somewhat later ascites is observed, which gradually increases until the abdomen becomes distended with fluid, with enlargement of the superficial abdominal veins. Meanwhile nutrition suffers sadly from disturbed digestion, from toxæmia due to hepatic inadequacy, and from interference with absorption. Though decided jaundice is rare apart from intercurrent duodenal catarrh, yet moderate yellowish tinging of the body-surface is often observed. In some cases we meet with more serious gastro-intestinal disturbance—nausea and vomiting, flatulence, and intractable diarrhœa. The urine is scanty, high-coloured, and deposits abundant urates. If the abdomen is not too distended, the enlargement and irregularities of the liver may be ascertained by physical examination, together with enlargement of the spleen. The legs may be œdematous. Toxæmic symptoms, closely resembling those of uræmia, may occur at any time, but more often in the later stages of the disease. Intercurrent attacks of moderate pyrexia are not uncommon.

Occasionally portal cirrhosis runs a rapid course of toxæmic type; but more often, partly as the result of the establishment of collateral circulation, and partly of compensatory hyperplasia of liver-cells, the course extends over two or three years; while sometimes the process undergoes temporary arrest, and the patient lives longer still. The occurrence of ascites usually marks the beginning of the end, and repeated tapping becomes necessary, until the patient dies from exhaustion. Sometimes, however, the fluid does not return, but the patient gets thinner and weaker, and dies from asthenia, or some secondary infection, especially tuberculosis and peritonitis,

In the earlier stages of portal cirrhosis, much benefit may be expected from *treatment*, and arrest of the disease may sometimes be secured; but later treatment can only be palliative. The chief indication is entire abstinence from alcohol in every form, and from stimulating food of every kind. The diet should as far as possible be limited to milk for several weeks. Alternate meals of Vichy water and warm skimmed milk (equal parts of each), and some light cereal food are recommended. Afterwards, eggs, light puddings, simple savouries of cheese and vegetable, and stewed fruit may be permitted, but no meat should be taken until convalescence is well established, and then only sparingly. With regard to drugs, the alkaline bicarbonates, with potassium iodide, or ammonium chloride are useful; but, in some cases, dilute mineral acids are preferable. The bowels should be kept freely open; and, for this purpose, saline aperients are desirable, with an occasional dose of calomel or blue pill at bedtime. The patient should be kept in bed to begin with; but, later on, gentle exercise may be allowed. When ascites causes much discomfort, or embarrassment of the breathing or circulation, paracentesis may be performed, and repeated as required. Under these circumstances, a pill containing mercury, squill, and digitalis, is a formula of some value. Hæmatemesis should be treated on the same lines as in ulcer of the stomach.

BILIARY CIRRHOSIS.

The exact nature of biliary cirrhosis is unknown, but it is probably due to inflammation of the smaller bile-ducts set up by a general infection of some kind, the toxins of which reach the liver in the general circulation. It is characterised by very marked uniform enlargement of the liver, accompanied with *intra*-lobular overgrowth of connective tissue, and jaundice. Microscopically,

besides the interstitial fibrous overgrowth, there is evidence of hyperplasia of liver-cells, and free formation of new biliary canaliculi. The spleen and abdominal lymph glands are also enlarged; and all the organs and tissues of the body are deeply stained with bile. The disease is most common in males of early adult age.

The onset of the disease is quite gradual. After a variable period of general malaise, weakness, and loss of flesh, jaundice appears, and becomes well-marked and permanent, though it fluctuates in intensity. The abdomen is prominent, especially in the upper part, from enlargement of the liver and spleen, but there is no ascites. The stools are dark-coloured, showing that the entry of bile into the bowel is not obstructed. There are periodic attacks of pyrexia, usually moderate but sometimes severe and attended with nausea and vomiting, or serious nervous disturbance. At such times there may be considerable abdominal pain, increase of the jaundice and leucocytosis. On physical examination, the liver is greatly enlarged, firm, smooth, and presenting a well-defined lower edge. The spleen is also enlarged, more so indeed than in portal cirrhosis. The general health steadily fails, and the patient ultimately dies from intercurrent complications, or grave toxæmia. The average duration is four or five years. Only palliative treatment is available.

Note on so-called Syphilitic Cirrhosis.—Syphilitic lesions of the liver may be either *inherited* or *acquired*. As a result of congenital syphilis, the liver and spleen may be enlarged and fibrosed as the result of diffuse chronic inflammation. In acquired cases gummata may form in the liver, which necrose at their centres, and form the starting-points of bands of cicatricial tissue. As the result of subsequent contraction of these fibrous bands deformities of the liver are apt to occur. Under these

circumstances, ascites may develop, and lead to confusion with portal cirrhosis; but the changes are often confined to one lobe of the liver, there is far less disturbance of the general health, jaundice seldom occurs, and the condition is amenable to anti-syphilitic treatment.

PERIHEPATITIS.

Perihepatitis, or inflammation of the peritoneal covering of the liver, may be acute or chronic. The acute form is more common in tropical regions, as a secondary result of acute congestion or inflammation of the liver; and, as a local affection, it may arise from extension of inflammation from the pleura or pericardium. Its symptoms and treatment cannot be separated from the symptoms and treatment of the affection on which it depends.

Chronic perihepatitis may be local or general. The former may occur from pressure by tight clothing, or in association with chronic structural changes in the liver, but is of very little clinical importance. On the other hand, chronic general perihepatitis requires further consideration. It is not strictly a disease of the liver, but a local result of chronic peritonitis, dependent on some general toxic condition of obscure origin and nature. It is frequently associated with adhesive inflammation of pleura or pericardium, arterio-sclerosis, or granular kidney, due to the same cause.

It is characterised anatomically by a thick layer of firm, hard, fibrous tissue, closely investing the peritoneal covering of the liver—more particularly on its convexity, often contracting adhesion with the diaphragm and adjacent parts, and compressing the liver by shrinking upon it. The spleen is similarly involved. The liver is not cirrhotic, but soft and fatty. The gall-bladder is empty, and almost lost in the fibrous investment. More or less evidence of

diffuse peritonitis is present, in the shape of ascites, rolled-up omentum, or adhesion of adjacent coils of intestine. The affection is more common in males than in females, and in early middle life.

The onset is very gradual and the course very protracted. Ascites is its principal feature, and the general health is not seriously compromised till a late stage. The effused fluid is clear, with a moderately high sp. gr., due to the large proportion of albumen. It is of inflammatory origin in the main. When the omentum is shrunk and puckered it is often felt on palpation, but must not be confounded with the lower edge of the liver. Tapping the abdomen is much better borne than in ordinary cirrhosis, and may be repeated as often as the fluid returns. Constitutional disturbance is also far less, and jaundice never occurs. The prognosis is unfavourable, though the end may be long deferred. The treatment is purely symptomatic.

DEGENERATION OF THE LIVER.

FATTY LIVER.

The distinction between fatty degeneration and fatty infiltration, as applied to the liver, is somewhat artificial, since the two are so often associated with each other. Still, it is clinically convenient to do so. Fatty infiltration of the liver (which is but an exaggeration of its normal function) is met with in two very opposite conditions: (1) in large eaters and drinkers (especially of beer) who lead indolent lives; and (2) in diseases attended with rapid emaciation, especially in *phthisis*. The paradox lies only on the surface. In both, the blood is loaded with fatty matter, traceable in the first case to an excessive supply of materials capable of being converted into fat;

and in the second case to the patient's own tissues. The greater frequency of the affection in phthisis than in other wasting diseases may be explained by respiratory insufficiency, possibly coupled with toxæmic factors.

ANATOMICAL CHARACTERS.—The organ is enlarged and heavy, the margins are thick and rounded, the surface is smooth, the consistence is soft and doughy, and the surface on section is pale and greasy. *Microscopic examination* shows that the cells are enlarged, and loaded with fat-globules. The process *begins at the circumference of the lobules* and extends inwards.

SYMPTOMS.—There are no characteristic symptoms. The physical signs are—(a) smooth uniform hepatic enlargement; (b) soft rounded lower edge; (c) *no* pain, *no* ascites, and *no* jaundice.

ALBUMINOID OR LARDACEOUS LIVER.

As the above terms imply, this affection is a lardaceous degeneration of the hepatic tissue. It is met with especially as a result of long-standing suppuration, constitutional syphilis, or phthisis.

ANATOMICAL CHARACTERS.—The organ is enlarged and heavier than normal, the edges are round and thickened, the surface is smooth, and the consistence is firm and resistant; the surface on section is dry, glistening, and exhibits no trace of lobular structure, and the affected tissue responds to the chemical tests for lardaceous change before described (p. 24). *Microscopical examination* reveals the characteristic change in the minute arteries and capillaries of the hepatic lobules. The process begins in the *middle zone of the lobules* corresponding with the distribution of the hepatic artery.

There are no characteristic *symptoms*. The *physical signs* consist of smooth, uniform, hepatic enlargement, with firm rounded resistant lower edge. There is *no*

pain or tenderness; and when jaundice or ascites is present it is due to pressure on the bile-duct or portal vein respectively, caused by lardaceous lymphatic glands in the hilum of the liver. The implication of other organs with a similar deposit, notably the spleen, kidneys, and bowels, is of considerable diagnostic importance.

HYDATID TUMOUR OF THE LIVER.

This affection is due to the development in the liver of the *cysticercal embryo* of a tape-worm, known as *Tænia echinococcus*, which infests dogs and wolves. The relationship between a cysticercus and a fully developed tape-worm will be understood on reference to the description already given of intestinal worms. It will then be seen that, contrary to the usual rule, it is the asexual cysticercus in this case which inhabits man, while its corresponding *Tænia* is found elsewhere. The ova discharged by the dog in the usual way are introduced into the human body through the medium of water or food, and on arriving in the stomach the embryo is set free, and finding its way into the portal vessels, it is carried along and lodges in the liver as an embolus. Sheep are affected in a similar way, and it is probably through eating their flesh that dogs become the seat of this tape-worm.

A hydatid tumour consists of a primary sac or vesicle (mother-cyst) with thick soft transparent and laminated walls, the innermost layer of which is formed by a delicate cellular membrane. The cavity of the mother-cyst is filled with clear limpid fluid of low specific gravity, which contains a large amount of chloride of sodium, but *no* albumen. From the inner surface, small cystic buds project, surmounted by a head furnished with four suckers and a row of curved hooklets. These buds afterwards become detached and form daughter-cysts suspended in the

fluid of the primary sac. Another generation is formed in the same way within the daughter-cysts, and the process may be repeated to a third or fourth generation. The whole tumour thus constituted is enclosed by a zone of connective tissue of irritative origin. Only one such cyst may be present, which may attain an enormous size, but smaller multiple cysts are not uncommon.

The history of a hydatid cyst varies. Thus, it may die and shrivel up—sometimes in consequence of the escape of bile into it, or it may suppurate, or it may burst into the peritoneal cavity or other adjacent parts. Hydatid disease is most common between twenty and forty years of age.

SYMPTOMS AND SIGNS.—Some cases run an entirely latent course from first to last; in others, there may be the physical signs of hepatic tumour without obvious functional disturbance; in a third group, by reason of its size or position, the tumour may give rise to symptoms from pressure on adjacent parts, such as the diaphragm, portal vessels, or vena cava; or lastly, owing to suppuration of the cyst, the signs and symptoms may be those of hepatic abscess. The cyst sometimes ruptures into the peritoneum, stomach, colon, or pleura.

The usual physical signs are painless irregular enlargement of the liver, so that its natural shape is altered; occasional bulging in the hypochondrium, and fluctuation on palpation when the cyst is large and superficial. An exploratory puncture with a small trocar may clear up the diagnosis in doubtful cases.

TREATMENT.—Where practicable an attempt should be made to remove the fluid with a fine trocar and cannula, which may be employed either with or without aspiration. The removal even of a portion of the fluid has been sometimes found sufficient to cause the death of the parasite. If this fails, the cyst should be boldly incised, and its

contents evacuated. Medicinal treatment beyond that which may be directed to the relief of symptoms is of no service whatever.

CANCER OF THE LIVER.

Cancer of the liver is far more often *secondary* to cancer somewhere in the portal area, but it is occasionally *primary*. It is most common in middle or advanced life.

ANATOMICAL CHARACTERS.—Secondary cancer almost always occurs in the form of isolated rounded nodules, which are scattered through the organ, and vary greatly in size. Those which are superficial form flattened nodular projections which become depressed in the centre, and are generally associated with more or less local peritonitis. The nodules grow at the periphery, and at their centres soon undergo degeneration. The adjacent parts of the liver are compressed, and the liver cells more or less atrophied and degenerated. The gall-bladder is distended as a rule, and may contain gall-stones; and the glands in the hilum of the liver are enlarged.

Primary hepatic cancer sometimes takes the form of a diffuse infiltration, and the irregularity of surface, so characteristic of the nodular form, is wanting.

SYMPTOMS.—There may be general malaise, weakness and loss of flesh for a considerable time before the nature of the case is recognised; or, again, the earlier symptoms may be those of the primary lesion, such as cancer of the stomach or rectum. The first local symptom is usually discomfort or actual pain (sometimes severe) in the region of the liver, which is found to be enlarged, resistant, and often tender on pressure. Then, or later, irregularities on its surface may be detected by palpation. Local friction-sound, due to perihepatitis, may sometimes be heard. Jaundice and ascites are present in about

half of all cases, and when once developed do not disappear. These conditions are usually the result of pressure on the bile-duct and portal vein by enlarged glands in the portal fissure. The spleen is rarely enlarged. Constitutional weakness, anæmia, and emaciation steadily progress (*cancerous cachexia*), until the patient sinks from toxæmia, or exhaustion, or some complication. The duration from the first appearance of symptoms rarely exceeds six or eight months, and the termination is always fatal.

DIAGNOSIS.—In advanced cases the diagnosis is generally easy, but in the earlier stages it may be very difficult. From cirrhosis it may be distinguished by the greater prominence of local pain and tenderness, the greater frequency of marked jaundice, the less frequency of ascites, the evidence of cancer in other parts, the greater rapidity of its course, and by the history of the case. When jaundice has supervened, distinction is necessary from biliary cirrhosis and impacted gall-stones. In the former, the age-incidence is much earlier, the liver is smooth, ascites is absent, jaundice is not of the obstructive type, periodic pyrexia is usual, and the general health suffers but slowly. In the latter there is more often a history of biliary colic, the liver does not *progressively* enlarge, there are no irregularities on its surface, and the intensity of the jaundice is apt to fluctuate.

The *treatment* is necessarily purely palliative and symptomatic.

TUBERCULOSIS OF THE LIVER.

This affection is never primary, and is only met with as part of general tuberculosis, or as secondary to chronic tubercle elsewhere. It cannot be recognised during life.

JAUNDICE.

JAUNDICE or ICTERUS is only a *symptom*, yet it is one of sufficient importance to require a separate description. Jaundice denotes a yellow staining of the skin and conjunctiva with bile-pigment due to the presence of bile in the blood. Various discolorations of the skin in some measure resemble that of jaundice, but they can always be distinguished by the absence of bile-pigment from the urine, and by the natural colour of the conjunctiva.

I. The commonest form of jaundice is that which depends upon obstruction in the bile-ducts external to the liver, and usually distinguished as *obstructive jaundice*. The explanation of it is that as the bile accumulates behind the obstruction, the bile-ducts become dilated, especially at their inter-cellular origin, and finally rupture into the lymph-spaces of the lobule, whence the bile is carried to the thoracic duct, and so reaches the blood-vessels. It may be caused (a) by the impaction of foreign bodies in the bile-duct, such as gall-stones; (b) by alteration in the walls of the duct from inflammation, stricture, or new growths; or (c) by pressure on the bile-duct from without, by enlarged glands, aneurism, or other tumours.

SYMPTOMS OF OBSTRUCTIVE JAUNDICE.—It will suffice to enumerate those symptoms which occur irrespectively of the cause of the jaundice. 1. *External discoloration*. As a result of impregnation with bile-pigment, the tissues soon become stained a yellowish or olive-green colour. This is well shown in the conjunctiva and skin. 2. The *urine* is also altered in colour, from the presence of bile and stains the linen. The colour varies from saffron-yellow to brownish-black according to the quantity of bile which it contains. The chemical tests for bile-pigment in the urine are as follows:—A drop or two of strong nitric

acid added to a small quantity of urine containing bile, on a piece of white porcelain, will give a play of colours due to progressive oxidation, passing through green, blue, violet, and red, into a dirty yellow. Also the addition of a few drops of a solution of iodine to urine containing bile, yields a green colour, and constitutes a very handy test. As regards other secretions, the sweat is bile-stained; but saliva, tears, mucus, and milk are seldom affected. 3. *The absence (partial or complete) of biliary colouring matter from the stools*, which are pale and clay-coloured and offensive. The pale colour is intensified by the presence of undigested fat. 4. *Gastro-intestinal derangements*:—Loss of appetite, gastric over-acidity, flatulence, and constipation. A bitter taste in the mouth is also sometimes complained of. 5. *Troublesome itching of the skin* is often observed, and *cutaneous eruptions* of various kinds are not infrequent. 6. The *temperature*, in the absence of any concurrent cause of fever, is slightly lower than normal. 7. The *pulse* is *slow*, and may fall to fifty, forty, or even twenty per minute. 8. *Impairment of general nutrition* is manifested by debility, emaciation, anæmia, and a not infrequent tendency to hæmorrhages from the various mucous membranes. 9. *Nervous symptoms*:—There is always some languor, depression, and incapacity for exertion; and sometimes more severe nervous disturbance occurs, which is probably due to acid-intoxication from interference with the detoxifying function of the liver.

II. Jaundice is also observed in another group of cases, in which there is no obstruction of the external bile-ducts, and hence often described as *non-obstructive*. This term should now be discarded, as it seems probable that, after all, an obstructive factor does enter into the causation of all such cases, though it mainly affects the bile-ducts *within* the liver. Since this type of jaundice is invariably

associated with toxic conditions, it is better to call it *toxæmic jaundice*. It may be caused (a) by poisons, such as toluylendiamin, and phosphorus; (b) by infections, such as yellow fever, pneumonia, and various specific fevers; and (c) by special forms of toxæmia (as yet not understood) as in acute yellow atrophy of the liver, and malignant jaundice.

The pathology of toxæmic jaundice has been variously interpreted; but, according to Dr. William Hunter, the chief factors are (a) destruction of the red cells of the blood (*hæmolysis*), causing an increase of effete hæmoglobin from which bile-pigment is derived; (b) a viscous condition of the bile; and (c) a concurrent catarrh of the intra-hepatic bile-ducts, which in some degree hinders the escape of bile from the liver.

SYMPTOMS OF TOXÆMIC JAUNDICE.—As compared with obstructive jaundice the chief differences are:—the presence of bile in the stools; the less intensity of the jaundice—as a rule; and the much greater liability to constitutional and grave nervous disturbance.

DIAGNOSIS.—The diagnosis of the condition upon which any given case of jaundice depends may be very easy, but it is sometimes very difficult, and hasty conclusions are dangerous. The matter cannot be fully discussed here, but the chief points to which attention should be paid are the following:—the presence or absence of bile in the stools; the presence or absence of fever; the onset, evolution, and duration of the jaundice; the presence or absence of pain—and its behaviour if present; the condition of the liver and gall-bladder, as ascertained by physical examination; the state of nutrition; together with the age and habits of the patient. Ascites associated with jaundice in an elderly subject almost always points to malignant disease. One word also of caution against forming too favourable an opinion of any case of jaundice which lasts longer than a month.

ICTERUS NEONATORUM.

There are two chief varieties. 1. A mild form, appearing on the first or second day after birth. The yellow colour curiously affects the skin before the conjunctivæ; and sometimes the latter remain unstained. The urine is only slightly tinged, and the stools are of normal colour. There is no disturbance of the general health, and the discoloration passes off in about a week. Its cause is obscure; but it is possibly related to an exaggeration of the hæmolysis which normally occurs in the first few days of life. 2. A severe and generally fatal form of a febrile septic nature, most frequently associated with umbilical infection.

ACUTE ATROPHY OF THE LIVER—MALIGNANT JAUNDICE.

This somewhat rare disease is most common in pregnant females, and rarely occurs in childhood or after thirty years of age. It is probably of a toxic nature. It is characterised by rapid atrophy and disintegration of the secreting tissue of the liver.

SYMPTOMS.—During the first few days there is nothing to distinguish the case from ordinary catarrhal jaundice; but then there is a sudden change for the worse. The patient becomes violently sick, the jaundice deepens in colour, and grave nervous symptoms supervene, such as severe headache, intolerance of light, delirium, muscular twitching or convulsion, which pass on to coma. There is a striking liability to *hæmorrhage*, indicated by petechiæ on the skin, epistaxis, hæmatemesis, and melæna. The temperature is more often sub-normal, but is apt to rise abruptly just before death. As the bowels are generally confined, and the stools so apt to be dark-stained from the presence of blood, conclusions as to the presence or absence of bile are attended with difficulty; but as the

formation of bile is greatly diminished or suspended in the later stages, little can reach the bowel. The urine is bile-stained, urea is greatly diminished, leucin and tyrosin are generally present, and albuminuria is not infrequent. Tenderness in the neighbourhood of the epigastrium is often complained of. Physical examination reveals a *rapidly progressive diminution in the size of the liver*. The disease is almost always fatal, and death usually takes place within a week. Occasionally, however, the duration is more protracted. When it arises during pregnancy, abortion or miscarriage usually follows.

Treatment is generally of little avail, and must be necessarily symptomatic. Smart purgation has been recommended.

SUB-SECTION VII.

DISEASES OF THE GALL-BLADDER AND BILE-DUCTS.

ACUTE CATARRHAL CHOLANGITIS. CATARRHAL JAUNDICE.

Catarrh of the common bile-duct is of frequent occurrence, and is usually due to extension of inflammation from the duodenum along the common bile-duct. It is met with most frequently in the young, but it may occur at any age. Errors of diet, exposure to cold, and alcoholic excess are its commonest causes. It may also occur in specific fevers, and occasionally in an epidemic form. The mucous lining of the duct is probably swollen, and the lumen is further obstructed by mucus.

The *symptoms* are those of catarrhal gastritis coupled with obstructive jaundice. There is usually some fever, at least at the onset; but as a rule it is inconspicuous. The liver may be a little enlarged and tender from hepatic congestion. After about a week the gastric symptoms

subside as a rule. Somewhat later, bile begins to reappear in the stools, the skin becomes clearer, and recovery usually takes place in the course of two to four weeks. More extended duration should arouse suspicion that the case is not one of simple catarrh. The diagnosis mainly turns on the exclusion of other causes of obstructive jaundice, and the youthful age of the patient.

More chronic forms of biliary catarrh are met with as a secondary result of gall-stones, or cancer of the liver.

As to *treatment*, an initial dose of calomel is desirable, and the bowels should afterwards be kept open by saline aperients or confection of sulphur. The patient should be kept in bed, on simple skimmed milk, until the jaundice disappears. It is often wise to give nothing for the first day or two beyond free drinks of water, Vichy water, or imperial drink. Return to ordinary diet should be gradual. A very useful medicine is bismuth in combination with salicylate of soda. Full enemata of warm water have been recommended.

Chronic catarrh of the bile-passages is a common result of gall-stones, and malignant disease.

SUPPURATIVE CHOLANGITIS.

This somewhat uncommon disease is due to infection which reaches the bile-passages, either by the portal or general circulation, or possibly, occasionally, by the common bile-duct. It is doubtful whether it ever supervenes upon healthy bile-passages—chronic catarrh is the commonest antecedent; and the organisms most often concerned are the *B. coli*, and the *B. typhosus*.

When the gall-bladder alone is affected, it forms a rounded tumour projecting below the edge of the liver, attended with pain and tenderness due to local peritonitis. If recognised early, surgical interference may sometimes be successful. When suppuration occurs with obstruction

in the common bile-duct, the condition is much more serious. The bladder and ducts (both outside and inside the liver) become distended with pus. The liver is enlarged and tender, and jaundice is always present. There is also fever of a hectic type, with rigors and profuse perspiration, and rapid loss of flesh and strength. Surgical interference sometimes yields substantial relief, but more often the case ends fatally.

GALL-STONES. CHOLELITHIASIS.

Gall-stones are formed in the gall-bladder, and may remain there without causing any symptoms. On the other hand, they may pass into the bile-ducts and cause severe paroxysmal pain—*hepatic* or *biliary colic*; or, they may excite inflammation, suppuration, or perforation of the gall-bladder; or, lastly, they may become impacted in some part of the bile-ducts. Gall-stones are rarely met with under forty years of age, and are most common in women of sedentary habits and prone to rich feeding.

Gall-stones may be single, but are more often multiple, and sometimes extremely numerous. Their size varies inversely to their number—from mere grains to the size of a pigeon's egg. They are generally rounded in form; but when many gall-stones develop simultaneously, their opposed surfaces become flattened against each other. Taken together, the tendency is to form a mould of the cavity which they occupy. Their surfaces are commonly smooth, but sometimes tubercular; and they vary in colour from a pale yellow to a deep brownish-black. When fresh they sink in water. Their consistence varies also, but they are usually soft enough to be cut with a knife. They consist mainly of cholesterine impregnated with bilirubin and lime salts. So-called "*biliary sand*" occurs in the form of hard, coarse, brownish-black grains of irregu-

lar shape, and consists of bilirubin combined with lime salts, without any cholesterine. It is now generally held that bilirubin and lime are precipitated from the bile by bacterial growth, and that cholesterine is a disintegration product derived from the mucous cells lining the gall-bladder.

SYMPTOMS.—There is a tendency to associate gall-stones far too closely with the occurrence of hepatic colic; so that, in the absence of colic, the true interpretation of other symptoms due to gall-stones is liable to be misread. More or less inflammation of the gall-bladder (*cholecystitis*) accompanies the majority of cases of gall-stone, but so long as there is free drainage through the cystic duct, there are no symptoms. When, however, drainage becomes hindered by swelling of the mucous lining of the duct, or by obstruction due to a plug of inspissated mucus or a stone at its upper orifice, some accumulation of the inflammatory exudate takes place; and gives rise to discomfort or pain—more often without jaundice or colic. Probably many attacks of so-called “indigestion” occurring independently of food, and without obvious cause, are really due to gall-stones, with imperfect drainage of the gall-bladder. With more complete obstruction, exudation accumulates to such a degree, that in conjunction with spasm of the gall-bladder, a stone may be forced into and passed along the duct, giving rise to the severe colic which characterises a typical attack of hepatic colic. Closely similar attacks may occur from obstruction of an inflamed gall-bladder, apart from the passage of gall-stones.

Hepatic Colic.—The onset is sudden, and marked by agonising pain in the region of the liver which radiates chiefly upwards to the right side, back, and shoulder. It is often accompanied with more or less fever, nausea, and vomiting. The general condition is one of profound prostration, and the violence of the pain may induce serious

nervous disturbance. The region of the gall-bladder is tender on pressure, and the latter may often be felt on palpation. In long-standing cases, as the result of inflammatory change, it becomes thickened and shrunken, so that it can no longer be felt. After a duration, which varies from one to several hours, the attack ends more or less abruptly, leaving the patient very prostrate, with slight fever, and dull aching in the abdomen. If the attack is at all protracted, obstructive jaundice follows. Other attacks, milder as a rule, are apt to recur at variable intervals. The gall-stone should be searched for in the stools, by diluting the latter with water, and straining through a sieve:

Impaction.—Instead of escaping into the bowel, a gall-stone may be arrested in the common bile-duct, generally in its dilated lower end (ampulla of Vater). Two results follow, obstructive jaundice and secondary infective inflammation of the bile-ducts. Though the obstruction is persistent, it is seldom complete, and as bile accumulates in the ampulla the latter dilates and permits the passage of more or less bile into the bowel. For this reason the intensity of the jaundice fluctuates, with corresponding variations in the colour of the stools. The liver is often moderately enlarged, and tender on pressure, as the result of cholangitis; and later on, it tends to become somewhat hard from increased formation of connective tissue. As a rule the gall-bladder is shrunken and cannot be felt. Ague-like paroxysms, due to acute exacerbations of local infection, accompanied with chill, fever, and sweating, are very characteristic; and sometimes there is more or less persistent fever.

Among the rarer effects of gall-stones are *suppurative inflammation*, *ulceration*, and even *perforation* of the gall-bladder, so that a large stone may pass directly into the duodenum or stomach. In other cases gall-stones

may be associated with cancer of the gall-bladder and ducts.

The prognosis of uncomplicated hepatic colic is favourable as a rule, but it is sometimes fatal. Impaction and suppurative inflammation are always serious complications.

TREATMENT.—During the passage of a gall-stone, the pain is best relieved by the hypodermic injection of morphia combined with a full dose of atropia (m.j.-ij.) repeated as necessary. In very severe cases it may be necessary to induce anæsthesia by chloroform. Hot fomentations should be applied to the abdomen, or a hot bath may be given.

Between the attacks much benefit may sometimes be derived from drachm doses of phosphate of soda, or Carlsbad salts in hot water in the early morning before breakfast, and a glass of hot water (plain) before the later meals. The success with which gall-stones have been removed by abdominal section justifies the adoption of surgical measures in all cases of frequently repeated biliary colic; and for impaction it is the only treatment available.

CANCER OF THE GALL-BLADDER.

CANCER of the gall-bladder is almost invariably primary; and, in a large majority of cases, is secondary to cholecystitis, associated with gall-stones. It is of a cylindrical or cuboidal cell type, and is much more common in women than in men.

The *symptoms* are often preceded by a history of hepatic colic, or evidence of cholecystitis. The onset is very insidious; and, in the early stages, is generally limited to vague dyspeptic disturbance, with possibly some local tenderness. The earliest physical signs are those of simple distension of the gall-bladder; and there is nothing which is characteristic unless a nodular tumour can be detected.

Jaundice occurs in three-quarters of all cases, and is usually due to extension of the growth to the bile-ducts, causing obstruction to the flow of bile. The general health is progressively and rapidly impaired, and death from exhaustion or hepatic toxæmia takes place within a year of the onset, at most. Apart from a local nodular tumour, the condition cannot be clinically distinguished from cancer of the head of the pancreas.

SUB-SECTION VIII.

DISEASES OF THE PANCREAS.

GENERAL CONSIDERATIONS.

The functions of the pancreas are partly digestive, and partly metabolic. Impairment of digestive function is indicated by general malnutrition, and alteration in the stools. The latter are bulky from increased undigested residue, pale and greasy from the presence of undigested fat, with a peculiar and offensive odour. Impaired metabolism is indicated by glycosuria. These functions, being attached to the secretion of different cellular structures, may be affected together or separately. Long-continued pancreatic disease is sometimes associated with a hæmorrhagic tendency. Also, a condition known as *fat-necrosis* occurs when, from any cause, pancreatic secretion escapes into the tissues; and is therefore most often met with in the neighbourhood of the pancreas. It gives rise to tallowy-white spots or patches of variable size. It is due to the action of a fat-splitting ferment in the pancreatic secretion on the fat-cells of the omentum, for instance, and combination of the liberated fatty acids with calcium (soap), which is deposited in the cells. It is most common in hæmorrhagic pancreatitis.

ACUTE HÆMORRHAGIC PANCREATITIS.

The condition is fortunately uncommon, for it is almost invariably fatal. Little is definitely known as to the causes that give rise to it. In certain cases it appears to be of traumatic origin; and in others, association with impaction of a biliary or pancreatic calculus has been noted. . The organ is enlarged, and on section shows numerous hæmorrhages. Hæmorrhage may also extend into the surrounding tissue, or lesser peritoneal sac. This is followed by widespread necrotic changes in the gland. In cases in which death is deferred for sufficient time, the gland may become gangrenous.

The affection is more common in men, between 25 and 50 years of age. The patient is suddenly seized with intense pain in the epigastrium, which is accompanied with severe vomiting, profound shock, and abdominal rigidity. Occasionally a history of previous attacks of epigastric discomfort or biliary colic may be obtained; but, as often as not, health is apparently normal up to the moment of onset. Later on, the abdomen becomes distended, and satisfactory palpation is rarely possible. The bowels are more often costive, and there is little or no fever. The symptoms are unrelieved, and death usually occurs in the course of a few days. Should the patient survive longer, and gangrene supervene, the temperature rises, and constipation is replaced by diarrhœa. . Jaundice is sometimes observed, and not infrequently a tumour-like resistance may be felt above the umbilicus, due to hæmorrhage and inflammatory effusion into the small peritoneal sac. The conditions with which confusion is most likely to arise are acute intestinal obstruction, perforative ulcer of the stomach, and possibly biliary colic.

Nothing can be done in the way of treatment beyond the ordinary measures for the relief of pain.

CHRONIC PANCREATITIS.

Chronic pancreatitis can seldom be diagnosed during life. It is characterised by interstitial fibrosis of the organ, and the formation of cysts or calculi in its ducts. The affection may be suspected when we meet with pain and tenderness in the region of the pancreas, in association with glycosuria and wasting. In some cases free fat may be observed in the stools, and the latter are apt to be bulky in proportion to the food which is taken.

CANCER OF THE PANCREAS.

Cancer of the pancreas usually occurs in the head of that organ, and is not very uncommon. A variable degree of aching discomfort in the region of the organ is generally an early symptom, radiating through to the back ; but severe pain is not a prominent feature. It is accompanied by loss of appetite, and more or less digestive disturbance. Constipation is more common than diarrhœa. A palpable tumour is present only in a minority of cases. Marked and persistent jaundice is the rule, and it is sometimes associated with ascites, from pressure on the bile duct and portal vein respectively. The gall-bladder is generally distended. Glycosuria is also not infrequently observed.

The course of the disease is rapid, and the termination always fatal.

SECTION IX.

DISEASES OF THE URINARY SYSTEM.

THE URINE.

RENAL DISEASE and modifications in the characters of the urinary secretion are so intimately connected with each other, that a short account of the urine in health and disease is absolutely necessary.

URINE IN HEALTH.—Healthy urine is a clear watery amber-coloured fluid, with a specific gravity of about 1020. The reaction is acid, and mainly due to acid phosphate of soda. The average amount passed in the twenty-four hours is about fifty ounces in an adult, but it varies within wide limits according to the amount of fluid drunk, and the amount of water which escapes by other excretory channels. This amount contains about 400 grains of urea, about 10–12 grains of uric acid, and still less hippuric acid, kreatin, and kreatinine. In addition it contains a considerable amount of chlorides, phosphates, and sulphates, of which the bases are soda, potash, lime, and magnesia, together with a minute quantity of oxalic and lactic acids.

On standing, it undergoes certain fermentative and putrefactive changes. At first the urine becomes more acid, a result which is due to acid fermentation of mucus, and possibly other organic ingredients; but after a variable period it becomes alkaline from the conversion of urea into carbonate of ammonia, cloudy, and emits an offensive and characteristic odour.

URINE IN DISEASE.—*Physical Characters.*—The urine is always scanty in febrile conditions, acute renal inflammation, or renal congestion; and in serious destructive renal disease, or as a result of certain reflex influences such as occasionally happens from the passing of a catheter it may be suppressed. On the other hand, it is abundant in diabetes, in some forms of chronic renal disease, and as a sequel of certain paroxysmal nervous affections. The specific gravity, as a rule, is in inverse relation to the quantity of urine, but sometimes we find a high specific gravity with abundant urine, as in diabetes. The reaction is nearly always acid except in certain forms of dyspepsia, or where alkaline salts are taken in large quantities, or in vesical inflammation attended with premature decomposition of urine. If the urine be turbid when passed, it is due either to the presence of earthy phosphates, mucus, pus, or fat (chyluria). In the first case, the turbidity clears up at once on the addition of an acid. Pus may be distinguished from mucus by the presence of albumen in the former. Both pus and fat-globules can be detected with the microscope.

Urea is derived from destructive metamorphosis of the nitrogenous elements of the food and tissues. It is increased when food is taken in excess, in fevers, and in diabetes; it is diminished on the other hand in all cachectic conditions, in diseases of the liver attended with destruction of hepatic substance, and in certain organic diseases of the kidney. A serious diminution in the excretion of urea, occurring in the course of Bright's disease, usually portends an attack of uræmia. The quantity is best ascertained by an apparatus (of which there are several cheap and efficient forms) based on the principle of the decomposition of the urea by hypobromite of soda, and measurement of the nitrogen evolved.

Uric acid is an end-product of the metabolism of nuclein-

proteids. According to Sir W. Roberts, it exists in the urine as a soluble quadriurate, composed of a molecule of uric acid in union with one of a biurate. It is readily decomposed into its constituent parts under *certain conditions*, depositing the uric acid in the form of reddish-brown rhombic crystals. These conditions are high acidity, poverty in saline ingredients, and deficient pigment. The presence of such a deposit does not indicate an excess of uric acid. The quantity of uric acid in the urine is diminished in gout, and in most chronic diseases. It is increased in fevers, leucocythæmia, pernicious anæmia, and food which is rich in xanthin bases.

Urates of ammonia and soda occur as an amorphous deposit in concentrated urines. Similar uratic deposits are also a common result of hepatic, catarrhal, or febrile derangement. They are readily dissolved by heat or the addition of an alkali.

Leucin, tyrosin, and cystin are exceptionally observed in the urine of disease, but the reader is referred to special text-books on the subject for their characters and the conditions under which they are found.

Salts.—The most important salts from a clinical point of view are the phosphates and oxalate of lime.

Phosphates.—There are two classes of phosphates, (a) the alkaline phosphates of sodium and potassium, and (b) the earthy phosphates of lime and magnesium. The former are twice as abundant as the latter, and are always held in solution. Turbidity due to deposit of an amorphous phosphate of lime is a common occurrence in neutral or alkaline urine, and should not be interpreted as indicating excessive excretion of phosphates. It may therefore be met with as a result of free vegetable diet, in gastric over-acidity, or marked debility. It is promptly soluble on the addition of a few drops of acetic acid. On the other hand, increased phosphatic excretion may occur, quite apart

from such a deposit, as in certain forms of neurasthenia. Under somewhat obscure conditions, phosphate of lime is sometimes deposited as crystalline rods or needles, singly or grouped in rosettes. In urine which has undergone ammoniacal decomposition, either within or outside the body, ammonio-magnesian or triple phosphates are thrown down in the form of three-sided prisms with bevelled ends.

Oxalate of lime, in small amount, is a constant ingredient of normal urine; but, on account of its small solubility, a trifling increase in the acidity of the urine, or any excess in its supply by taking such foods as rhubarb or spinach, may lead to its deposit in a crystalline form, and cause considerable irritation of the urinary passages, or actual concretions. It crystallises in small octohedra, or (more rarely) in dumb-bell forms. It is derived mainly from food; but to a small extent, it is of endogenous origin. That which is ingested is broken up for the most part in the alimentary canal; but when taken in excess, and in certain forms of gouty dyspepsia, it appears as a deposit in the urine (*oxaluria*).

Indican, or indoxylsulphate of potassium, is derived from indol, which is a product of the decomposition of proteids in the bowel, and gives the characteristic odour to feces. It is of some importance as a rough guide to the amount of putrefaction going on in the bowel. The best test is to mix equal parts of urine and strong hydrochloric acid, adding a particle of bleaching powder. If indican be present, a blue colour appears on gentle agitation.

Melanuria, due to the excretion of melanin, is a certain sign of melanotic growths in the body. The colour of the urine is normal when first passed, but blackens on exposure. The same thing often happens from absorption and excretion of carbolic acid, creasote, naphthalene, or salol, but this may always be distinguished from true

melanuria by immediate blackening on the addition of nitric acid in the latter case. This does not occur with other forms of darkening.

Albumen in the urine is almost always of pathological significance. It is found under one of four conditions: (a) renal congestion; (b) structural changes in the kidney; (c) certain febrile and toxic conditions; and (d) the presence of blood or pus in the urine. The tests for albumen are based on the fact of its coagulation by heat or reagents. If urine containing albumen be boiled it becomes turbid, or a dense precipitate is thrown down (according to the amount present), which is *not* redissolved on the addition of a few drops of acetic acid. Again, if a little urine containing albumen be poured gently down the side of a test-tube containing strong nitric acid, a white layer of coagulated albumen forms at the line of contact between the two fluids. A saturated solution of salicyl-sulphonic acid is also a most satisfactory reagent, yielding a precipitate in the presence of the smallest trace of albumen. In forming an opinion as to whether albumen is the result of organic renal disease we are mainly guided by the amount and persistence of the albuminuria, and the associated presence of renal epithelium and casts.

Albumen is not infrequently found in the urine of adolescents who appear to enjoy good health, and in whom there is no other evidence of renal disease. The questionable term of "*physiological albuminuria*" is sometimes applied to such cases. After making due allowance for those cases in which albuminuria is the result of long antecedent renal disease thought to have been cured, and where it forms the initial symptom of organic renal disease as yet attended with no other symptom or sign, there still remains a certain proportion in which it is clearly due to functional causes. Of these the most important are exertion, nervous strain, and exposure to cold.

While we may admit the existence of albuminuria independently of organic renal disease, it is possible that some of these cases ultimately lead to such a result; and until we can discriminate functional from organic cases more surely than at present, insurance societies will probably continue to reject *all* cases of albuminuria. The main features of the functional variety are—small quantity, intermittent appearance, absence of tube-casts, normal heart, and limitation to the period of adolescence.

Sugar in the urine is almost invariably in the form of glucose or grape sugar, and hence the term *glycosuria*, which is applied to the condition. It may be temporary or persistent. Temporary glycosuria may occur from an excess of saccharine or carbohydrate food, in certain nervous affections, or after the administration of chloroform. It is also not uncommon in fat gouty subjects, but the line which separates temporary from persistent glycosuria (diabetes) is very ill-defined. The general characters of diabetic urine are described with that disease. The principal test for sugar is *Fehling's test*, which depends upon the reduction of cupric salts in hot alkaline solutions. The reagent contains cupric sulphate in solution with sodium potassium tartrate and caustic soda. As it does not keep indefinitely, it is better (when the test is seldom used) to have the copper salt in a separate solution, and to mix it as required. It is carried out by heating a little of the suspected urine with rather more than an equal bulk of Fehling's solution, when a reddish-yellow precipitate of suboxide of copper is thrown down if sugar be present. Urine sometimes contains copper-reducing substances in small quantity other than sugar, such as uric or glycuronic acid, or even lactic acid from administration of salicylates. It is well, therefore, to employ confirmation tests when the reaction is not well marked. Of these the best and simplest is the *fermentation test*.

A small quantity of yeast is added to a test-tube filled with the suspected urine, and the tube is then inverted over a vessel containing the same fluid, and allowed to stand in a warm place. As fermentation proceeds (if sugar be present) carbon-dioxide is given off, which collects in the upper part of the tube. The same test may be adapted for quantitative estimation. A more accurate quantitative test is afforded by using a standardised Fehling's solution, calculating from the quantity of urine necessary to decolorise a given measure of the copper solution.

Acetone, *aceto-acetic acid*, and *β -oxybutyric acid* are successive terms in a natural series of substances which may be found in the urine, especially in diabetes. Acetone may be present alone, but when *β -oxybutyric acid* is found, it is generally associated with the other two members of the group. Acetone is not confined to diabetes, but is often present in other conditions, attended with abnormal metabolism such as specific fevers, cancer, persistent vomiting, and certain toxic affections. When marked, the condition is known by the term "*acidosis*"; and diabetic coma is commonly attributed to this cause. It is believed to take its source mainly in an abnormal metabolism of fat, which is especially apt to occur when carbohydrate food is withheld or fails to be assimilated. Acetone gives an æthereal odour to the breath and urine when present in sufficient quantity.

On the addition of sodium nitro-prusside in solution to urine made alkaline by liquor potassæ, the further addition of acetic acid gives a dark violet colour if acetone be present (*Legal's test*). Ferric chloride gives a deep reddish-brown colour to urine containing aceto-acetic acid. Confusion may arise if the patient is taking any salicyl compound at the time. The detection of *β -oxybutyric acid* requires more elaborate methods.

Blood.—Blood in the urine (*hæmaturia*) may arise from hæmorrhage in any part of the urinary tract—(1) the kidney or its ducts; (2) the bladder; or (3) the urethra. In *renal hæmorrhage* the blood is uniformly mixed with the urine. The colour varies from dark-red like porter, to a smoky tinge, according to the quantity of blood present. There may be a dark-coloured sediment on standing, which may contain blood-casts. It is usually due to nephritis or more rarely to renal cancer. In free hæmorrhage from the kidney or its pelvis, long narrow clots (moulds of the ureter) may be found in the urine. In hæmorrhage from the *bladder* the blood is much less intimately mixed with the urine, and often appears only towards the *end* of micturition, and is associated with the symptoms of vesical disease. Hæmorrhage from the *urethra* is distinguished by its occurrence independently of micturition. Hæmorrhage may occur from any part of the urinary tract in purpura and other hæmorrhagic disorders.

The detection of blood corpuscles by microscopic examination of the urine is the safest test for blood. The following is the best test for blood-pigment:—Add some tincture of guaiacum to a small quantity of urine in a test-tube, and shake it up with a little ozonic ether. If hæmoglobin be present, the ether, which after a few moments collects on the surface, exhibits a blue colour. The appearance of the blood-cells varies according to the specific gravity of the urine; if it be high the cells are small and shrivelled, if it be low they are large, transparent, and spherical, in other cases the appearance may be normal. The urine is necessarily albuminous.

Cases also occur in which no corpuscles (or only a very few) are to be seen, and in which the discoloration is due to blood-pigment derived from breaking up of red blood-cells. (Refer to “Hæmoglobinuria.”)

Mucus.—The quantity varies from the slight cloud observed in healthy urine after standing, up to the viscid ropy urine of cystitis. Its amount is a measure of the irritation to which the vesical mucous membrane is exposed, and its presence in large quantity indicates actual inflammation. According to the intensity and duration of the inflammation, pus is intermixed with the mucus, in all degrees up to pure pus.

Pus.—Urine containing pus is turbid and does not clear on boiling, and can be readily recognised on microscopical examination. When present in quantity, pus forms a yellowish-white deposit, which becomes ropy on boiling with liquor potassæ. Pus in the urine may be derived from inflammation of any part of the urinary mucous tract, from renal abscess, or from direct communication with some neighbouring abscess.

Casts.—One of the most characteristic results of renal disease is the appearance in the urine of plastic moulds of the tubules, known as *casts*. They are of various kinds—hyaline, epithelial, granular, fatty, and hæmorrhagic.

Hyaline casts are delicate transparent structures, varying in size, according as they are formed in tubules retaining or stripped of their epithelium. They are the commonest and earliest indication of renal disorder. Their origin is not known for certain, and probably is not always the same. They may be due either to coagulation of exuded plasma, or to hyaline transformation of tubular epithelium, or to coagulation of some epithelial product. *Epithelial* casts are formed by the agglomeration of desquamated epithelial cells, or of other cells which replace them as disease proceeds. *Granular* casts are generally short, opaque, brownish in colour, and vary considerably in thickness. They probably arise from epithelial disintegration. *Fatty* casts are due to fatty

degeneration of epithelial casts; while *blood-casts* are obviously caused by coagulation of extravasated blood.

URÆMIA.

Uræmia is a clinical term applied to a rather ill-defined group of symptoms, almost certainly of toxic origin, which characterises the terminal stage of most cases of renal disease. Its immediate cause is unknown. Various theories have been advanced from time to time, but none are entirely satisfactory, and their consideration lies beyond the scope of our present purpose. It must suffice to say that, in general terms, uræmia depends upon the loss or suspension of renal function; and accordingly a marked diminution in the amount of urea excreted in the course of renal disease, is evidence of valuable diagnostic importance, as pointing to renal inadequacy, and thus often affording a warning of impending uræmia. Yet uræmia is not due to retention of urea, nor (so far as known) to retention of any ingredient of renal excretion.

The symptoms vary considerably in different cases, according to the intensity of the toxic process, to the rapidity of its development, and possibly also to variations in the nature of the toxic material. When uræmia develops slowly (*chronic uræmia*), and in the milder forms, some of the following symptoms are observed:—headache, muscular cramps or twitching, restlessness, drowsiness, occasional or inconstant delirium, nausea or vomiting, hiccough, diarrhœa, and itching of the skin. The breathing is often disturbed. Sometimes the dyspnœa is continuous, sometimes paroxysmal, or (more rarely) the breathing is of the Cheyne-Stokes type. It may be added that dyspnœa occurring in renal disease is not necessarily uræmic, but may be due to associated bronchitis and emphysema, pulmonary œdema, or cardiac failure. The special senses

may be affected, giving rise to dimness of sight, temporary blindness or deafness. In fatal cases drowsiness may deepen into coma; or convulsions may occur, followed by coma.

On the other hand it often happens that, without any warning, or after trifling premonitory disturbance, the patient is suddenly seized with convulsion of an epileptic type, several attacks sometimes occurring in rapid succession, or he may pass rapidly into a state of coma (*acute uræmia*). Uræmic convulsion is general as a rule, though often more marked on one side of the body. The pupils are dilated, and the knee-jerks are increased. Temporary paralysis—usually hæmiplegic—sometimes follows on convulsion; or, in rare cases, may occur independently of convulsion. Acute uræmia sometimes takes the form of sudden gastro-intestinal disturbance, sudden but temporary blindness, or (more rarely) of acute mania. In chronic uræmia, the temperature is usually sub-normal; but in severe convulsion it rises, and sometimes to a considerable extent.

Uræmia may be confounded with cerebral or meningeal hæmorrhage, epilepsy, alcoholic intoxication, or opium-poisoning. The *diagnosis* rests chiefly upon the previous history of the case, the presence of albumen and tube-casts in the urine, and association with those cardio-vascular changes which are so common in chronic Bright's disease.

The *prognosis* of uræmia in its more acute and violent manifestations is always grave, but by no means necessarily fatal. On the other hand, the liability to recurrence has to be reckoned with, and much depends on the extent of organic disease associated with it, and the response to treatment.

In *treating* uræmia, an endeavour should be made to reduce the formation of toxic products, and to encourage their elimination. The former indication is best met by

an exclusive milk diet, and the avoidance of all meat extracts; and the latter by dry-cupping followed by hot and moist applications over the loins, wet-packs, vapour-baths, and the internal administration of hydragogue cathartics, such as pulv. jalapæ co. Hypodermic injection of pilocarpine, beginning with a small dose (gr. $\frac{1}{10}$) and repeated as required, is sometimes useful as a prompt diaphoretic. It is not suitable for children. When convulsions supervene, an injection of chloral and potassium bromide may be given by the rectum. Venæsection is also well worthy of consideration under these circumstances, if not contra-indicated by the state of the heart and circulation. In profound coma, enemata of hot water, or subcutaneous injection of saline fluid may sometimes be resorted to with great advantage.

RENAL CONGESTION.

Congestion of the kidney may be either active or mechanical.

Active congestion of the kidney cannot be separated from acute nephritis, of which it forms the early stage.

Mechanical congestion generally depends upon venous obstruction from non-compensated dilatation of the heart. The kidneys are notably enlarged and engorged with blood; and, if the congestion is severe or long-continued, disintegration and degeneration of the renal epithelium may follow, with over-growth of the interstitial connective tissue. The urine is scanty and high-coloured. A moderate amount of albumen is usually present. If the amount be large, or if associated with renal epithelium and casts, the kidneys must be assumed to have undergone some structural change.

The general points to be observed in the *treatment* of renal congestion are absolute rest, a milk diet, warm and

moist applications or dry-cupping over the loin, and free purgation, combined with attention to the condition on which congestion depends.

BRIGHT'S DISEASE.

GENERAL CONSIDERATIONS.—Our knowledge of the group of renal diseases about to be described is not sufficiently exact and complete to admit of a rigid classification either on a clinical or pathological basis; so that, although the above heading is now used to cover a wider field than was known to Bright, it is preferred because it does not carry with it a committal to any particular theory as to the nature of the disease. Broadly speaking, the morbid conditions in question are characterised by inflammation of the kidney (*nephritis*). But there are some forms of Bright's disease, such as "granular kidney," which cannot be regarded as truly inflammatory, and again there are some forms of renal inflammation which, on clinical grounds, do not come within the four corners of what we call Bright's disease.

The chief causes of Bright's disease are specific infections, especially scarlet fever and diphtheria; toxic agents introduced from without, such as alcohol and lead; abnormal metabolic products generated within the body in connection with gout and chronic dyspeptic disturbance; and certain special conditions, such as pregnancy. Exposure to cold and damp undoubtedly ranks as an important contributory cause in some cases. The one factor common to all the above causes is *toxæmia*, under the influence of which the tissues of the kidneys become damaged. The precise form which such damage takes, and also its degree, depend upon the nature, virulence, and persistence of the toxic agent to which it is related. When the intoxication is intense and rapidly developed,

as in scarlet fever, an acute diffuse nephritis is the result. With less intensity and slower development a diffuse nephritis takes place which is chronic from the outset. On the other hand, with quite moderate but steadily persistent intoxication, we meet with atrophy and degeneration of renal epithelium, associated with interstitial fibrosis (*granular kidney*). Between the two extremes of acute inflammatory damage and non-inflammatory degeneration, there are intermediate forms, in which inflammatory and degenerative changes are variously combined. No doubt such differences are due in part to varying *qualities* of intoxication, but of these we know very little, and consequently are unable to measure their influence.

On the basis of these considerations, an acute and a chronic type of Bright's disease will be described, subdividing the latter into two varieties according as tubular or interstitial changes prevail. It must be understood that many intermediate types are met with in actual practice.

ACUTE BRIGHT'S DISEASE.

Acute Bright's disease is characterised by an acute diffuse nephritis. The kidneys are more or less enlarged, and engorged with blood; but the surfaces are smooth, and the capsules remain thin and non-adherent. On section the colour is at first of a deep dusky-red throughout, but at a later stage the cortex becomes paler or mottled, and contrasts strongly with the dark red bases of the pyramids.

Microscopical examination reveals engorgement of the glomeruli with nuclear proliferation of the glomerular epithelium; the capsular space is more or less filled with fibrinous coagula, enclosing leucocytes, red blood-cells, and granules; the convoluted tubules are choked with

epithelium in various stages of disintegration, leucocytes, blood, and tube-casts; and the interstitial tissue is often the seat of œdema and leucocytic infiltration.

While this description conveys a general idea of the structural changes, it should be understood that great irregularities are observed, both as regards their distribution and their incidence. Thus, areas of marked change are noted alongside others which are relatively normal. In scarlet fever, glomerular changes are apt to be more prominent. In diphtheria the tubules are most affected. Yet again, we find cases in which foci of interstitial œdema and infiltration are the chief lesion.

Symptoms.—Scarlatinal nephritis affords the most characteristic type of acute nephritis for the purpose of description. The onset is generally abrupt, and is sometimes marked by chilliness or (more rarely) by actual rigor, and sharp rise of temperature. More commonly pyrexia is slight and is often absent altogether. Frequently, œdema is the first symptom which attracts attention, sometimes even before albumen appears in the urine. It begins in the face, and rapidly spreads to other parts. Dropsy may be quite moderate, or extreme, and possibly attended with effusion into serous cavities. Changes in the urine quickly follow, if they have not already appeared. It is scanty, turbid, abnormally dark or smoky in colour, with a high specific gravity, and loaded with albumen. On standing, it deposits a copious sediment, which on microscopic examination is found to consist of blood-cells, loose renal epithelium, and abundant epithelial hyaline, granular, and blood-casts. Meanwhile, the patient may complain of aching across the loins, and pains in the limbs. The skin is markedly pale, dry, and often irritable; the tongue is coated; the appetite is lost; and the bowels are generally costive. The frequency of the pulse is increased at first, though it generally

slows down later, and the tension is raised. As the case proceeds, mild uræmic symptoms are of common occurrence, such as headache, mental dullness, nausea or vomiting, and sometimes diarrhœa.

With regard to *diagnosis* it should be remembered that acute Bright's disease occurring under other conditions often varies somewhat from the above description. Thus : the onset may be very insidious ; œdema may be very slight or absent ; or some acute uræmic trouble may be the earliest manifestation. Again, owing to the fact that in the acute nephritis of pregnancy the trouble falls almost entirely on the tubules of the kidney to the exclusion of the glomeruli, hæmaturia is uncommon. It is important clearly to differentiate acute Bright's disease from the albuminuria which so frequently accompanies the febrile stage of acute specific disorders.

The *progress* and *results* of acute Bright's disease vary greatly. In favourable cases (which constitute the majority) the symptoms begin to subside in the course of a week or thereabouts, the anasarca usually disappearing long before the albumen has quite vanished from the urine, and convalescence is gradually re-established. On the other hand, serious uræmic symptoms may supervene, such as convulsions or coma, and possibly prove fatal. Or again, complications, such as inflammation of the lung or pleura, or acute œdema of the lung or larynx may arise and prove fatal. Owing to the tendency to recurrence, great care should be taken until the albuminuria has quite disappeared.

Treatment.—The patient should be kept warm in bed at an equable temperature. In the early stages the bowels should be kept freely open by saline aperients in concentrated solution, or by compound jalap powder ; the action of the skin should be maintained by occasional warm water or vapour baths ; and dry cupping or hot

fomentations may be employed over the loins. Medicinally, full doses of liquor ammoniæ acetatis, well diluted, combined with digitalis and nitrous ether, are of service. As the more acute symptoms subside, vegetable tonics and iron are indicated. The best food is hot milk and Vichy water, which may be given freely. Alcohol is rarely desirable. Watery drinks may be freely allowed so far as to satisfy thirst. If the dropsy be so excessive as urgently to call for relief, the subcutaneous tissue may be drained by means of Southey's tubes.

CHRONIC BRIGHT'S DISEASE.

Chronic Bright's disease may occur as the sequel of an acute attack, but the vast majority of cases are chronic from the outset. The morbid changes may chiefly affect the tubular structures of the cortex, which, together with more or less fatty degeneration, bring about what is known as *chronic tubular* or *parenchymatous nephritis* or *smooth white kidney*; or may be mainly characterised by fibrosis, giving rise to *chronic interstitial nephritis* or *granular kidney*. The latter is always chronic from the beginning.

Though the changes in *chronic tubular nephritis* mainly affect the urinary tubules, yet it must be specially remembered that they are never limited to these, but always involve the interstitial tissue as well, to a greater or less extent. It is to differences in this respect that its anatomical varieties are due. In a typical case, the size and weight of the organ are greatly increased, its surface is smooth and pale, and the capsule is not adherent. On section the cortex is seen to be much thickened, pale or yellowish, thus contrasting with the darker pyramids. On microscopic examination, the tubes are found to be stuffed with epithelium in various stages of disintegration and often markedly fatty; while here and there they

contain fibrinous material. The glomeruli are large, the capsules are thickened, and the interstitial tissue is moderately increased.

The *granular* or *contracted kidney*, in its typical form, is small, its surface is granular or nodulated, and its capsule is firmly adherent. On section the cortex is found to be greatly reduced in thickness, hard and resistant, and sometimes studded with small cysts. In well-marked cases its colour is dark red, but in intermediate forms it may be greyish-red or mottled. On microscopic examination, the connective tissue is found in relative excess, especially round the glomeruli, which may be shrunken and fibroid. The tubules are variously altered—some are denuded of epithelium, others are dilated and lined by small cubical hyaline cells, some contain degenerated epithelium, and many are altogether destroyed. The renal arteries are greatly thickened. In all chronic forms of Bright's disease, but especially in granular kidney, cardio-vascular changes are very common, characterised by hypertrophy of the heart, and arterio-sclerosis.

SYMPTOMS.—Apart from those cases which supervene upon acute Bright's disease, the onset is gradual, and insidious. It may remain altogether unnoticed, until revealed by some acute uræmic manifestation. Among the earliest symptoms to attract attention are general malaise, dyspeptic troubles, slight œdema of the ankles towards the end of the day or puffiness of the eyelids in the morning, rising at night to pass urine, increasing pallor and failure of strength without anything obvious to account for it, a high-tension pulse, or visual troubles. The clinical characters, when the disease is established, vary according to the type of the disease.

Smooth White Kidney.—This type is for the most part characterised at first by progressive weakness, impaired nutrition, pallor, a pulse of high tension, puffiness of the

eyelids and ankles, loss of appetite, and frequent micturition. As the case proceeds, the pallor increases and dropsy becomes abundant. Dropsical effusions into the serous cavities may also occur. The urine is scanty, its specific gravity is normal or slightly increased, it is turbid or sometimes smoky, highly albuminous, deficient in urea, and on standing it deposits a copious sediment containing renal epithelium, leucocytes, sometimes blood, together with epithelial, fatty, granular, or hyaline tube-casts. Secondary internal inflammations are more common than uræmia in its more marked forms, but milder uræmic troubles such as headache, nausea or vomiting, and gastrointestinal disturbance are of frequent occurrence. Mucous hæmorrhages are not uncommon in the later stages, but marked cardio-vascular changes and retinitis are less common than in granular kidney. The above type is most common in middle life, less so in the young, and more frequent in males than in females.

Granular kidney is always chronic from the commencement and begins very insidiously, with symptoms such as those described with arterio-sclerosis (p. 324). The average age at which it occurs is forty to fifty years. The urine is copious, of low specific gravity, pale, clear, with very little albumen, and yielding little or no deposit on standing, but containing a few hyaline or granular casts, and some epithelium. Intermediate cases often occur in which the albumen is considerable, and casts are relatively abundant. Dropsy may be absent, and is generally slight and limited to some puffiness of the ankles. The face, though often pale, is not white or pasty. The skin as a whole is dry and harsh, and many cases exhibit progressive failure in nutrition. The pulse is almost always of high tension, and palpitation may be a marked symptom. Hypertrophy of the heart with evidence of arterio-sclerosis is very generally observed. Uræmic complications are common.

The disease is very chronic, and it may be altogether latent until the development of some serious complication, such as cerebral hæmorrhage, or acute uræmia, reveals its true nature. Other cases, again, may first seek advice for cardiac dilatation, relative mitral incompetence, and all the signs and symptoms of cardiac failure.

Chronic Bright's disease is incurable, in any case; but the course of the tubular is more rapidly unfavourable than that of the granular type, in which, with care, life may often be prolonged for several years.

The TREATMENT of chronic Bright's disease is not satisfactory. The body should be kept warm and be clothed in flannel. The action of the bowels and skin should be encouraged, and watery drinks should be liberally supplied. All exposure to cold must be carefully avoided, and residence in a warm equable climate is desirable. Milk, or milk supplemented by cereals, with vegetable purées should be given under ordinary circumstances. In any case meats of all kinds should be closely restricted, while soups and all forms of meat extracts should be avoided. An exclusive diet of milk (diluted) is desirable for a time, when there is any threatening of uræmia. Alcohol is not desirable as a rule, but sometimes digestion is carried on more efficiently with the aid of a little light wine, such as claret or hock. In the earlier stages no medicine is needed beyond simple aperients and saline diuretics. For anæmia iron is indicated. The tincture of the perchloride is the best preparation, but where this disagrees with digestion it must give place to the blander ammonia-citrate or tartrate of iron. If signs of cardiac dilatation supervene, digitalis in combination with strychnia is often of much service. Extreme dropsy is most effectually combated by purgatives and diaphoretics. Of the former, compound jalap powder, bitartrate of potash, elaterium, and gamboge are among the best; while the action of the

skin may be excited by warm baths or warm wet-sheet packs. Sometimes drainage of dropsical parts gives great relief, but from the readiness with which erysipelatous inflammation takes place, caution is necessary. The treatment of uræmia has already been alluded to, and secondary inflammations must be dealt with as they arise.

PYELITIS AND PYO-NEPHROSIS.

PYELITIS, or inflammation of the renal pelvis, is probably always of microbic origin. Among the many organisms which are found in this connection, colon and tubercle bacilli are of most importance. The channel of infection may be upwards from the bladder and ureter (*ascending infection*), or downwards from the general circulation by way of the kidneys (*descending infection*). As to the relative frequency of each there is great difference of opinion. It should be borne in mind that, since the urinary passages normally show great resistance to infection, accessory factors are generally necessary before infection becomes possible, such as feeble health, local injury from calculi or organic disease, or some obstruction to the out-flow of urine. One or both kidneys may be affected; but in *all* long-standing cases both kidneys tend to become involved. Though pyelitis is more common in adult life, yet children and infants are not infrequently affected—girls more often than boys.

MORBID ANATOMY.—As an acute condition it is characterised by redness, swelling, and ecchymosis of the lining mucous membrane, with shedding of its epithelium, and a discharge of muco-pus. In more chronic types the membrane is thickened, and sometimes becomes ulcerated or covered with a layer of phosphatic deposit. The opening into the ureter, already much narrowed by inflammation, may become blocked by calcareous particles, blood-clot,

or thickened pus, in which case the pelvis becomes dilated and filled with pus. Under these circumstances, suppurative inflammation may extend to the kidney (*pyelo-nephritis*); or the kidney may be converted into a multilocular sac filled with pus (*pyo-nephrosis*); or a communication may be established with the cellular tissue round the kidney, and set up a *peri-nephric abscess*, which burrows in various directions; or the whole mass may become inspissated and more or less calcified. In suppurative pyelo-nephritis dependent on cystitis (surgical kidney), the renal substance becomes acutely inflamed, with lines of pus between the tubules. Tuberculous pyelitis will be described separately.

SYMPTOMS.—In so far as the symptoms of pyelitis can be separated from those of the condition to which it is secondary, the following are the most important:—Aching pain in one or both loins, local tenderness, and frequent micturition, attended with more or less febrile disturbance of a hectic type. The urine is almost always acid unless the case is complicated with cystitis, turbid, and albuminous. The deposit, on microscopical examination, shows pus and blood-cells in variable quantity, together with elongated tailed or irregularly shaped epithelial cells. Moderate attacks of renal colic may occur from the passage down the ureter of blood-clot or purulent débris. If the ureter should become blocked—the other kidney being unaffected—the urine may for a time become normal, or contain only a trace of albumen; but should the obstacle give way, the urine returns to its former condition. Such a sequence of events may be repeated from time to time. Later on, a round tumour may be observed, due to pyo-nephrosis or peri-nephric abscesses, which is variable in size, tender on pressure, and obscurely fluctuating. Or the acute symptoms may subside with recovery; while, in a third group of cases, the condition becomes chronic.

In surgical kidney, over and above the signs of cystitis, severe pains in the back, shivering attacks with high temperature, and progressive toxæmia are observed.

The *diagnosis* of pyelitis in association with cystitis is often extremely difficult. Indeed, in the absence of symptoms referred to the loin, it may be impossible. It must not be forgotten that when the ureter is blocked the urine may be quite normal for a time.

The *prognosis* of pyelitis mainly turns on the nature of the condition to which it is due, and on the occurrence of complications such as pyo-nephrosis or peri-nephric abscess. Acute simple cases generally end favourably; and many chronic cases may do well for a long time, without serious results. Also where pus has formed, provided only one kidney is affected and drainage is free, the general health suffers but little; but, under contrary conditions, the prognosis is more grave.

The *treatment* for the most part consists in ascertaining and removing the cause when this is possible. In acute cases, rest in bed, simple unirritating food, and citrate of potash or soda in sufficient doses to make the urine alkaline are indicated. Poultices or fomentations to the loin may be employed to relieve the pain. When the disease has become chronic, drugs are of little service. If the trouble can be localised to one kidney, with the presumption that the other is unaffected, and also if the diagnosis of a pyo-nephrosis has been established, surgical exploration is called for, with a view to the removal of the kidney.

PERI-NEPHRIC ABSCESS.

Suppuration in the connective tissue which surrounds the kidney may occur from traumatic injury of the loin, or secondarily to disease elsewhere, such as extension

from suppurative appendicitis, or a pelvic abscess, or pyæmic infection. It is probably most common as the result of suppurative pyelitis, with or without perforation. As a rule it gives rise to a *definite tumour* in the loin. The abscess, if not interfered with, may burrow in various directions; or it may point in the loin posteriorly, or at Poupart's ligament. In the absence of a tumour, the signs are equivocal. The earliest indications consist of *pain* and *tenderness*, which are most marked in the back, between the last rib and the pelvis. The pain may radiate into the leg of the same side, sometimes accompanied with slight puffiness of the foot and ankle. The leg on the affected side may also be sometimes flexed on the trunk, and cannot be straightened without causing pain. There is also generally more or less *fever* and constitutional disturbance of a hectic type. The chief conditions from which it has to be distinguished are appendicitis, psoas abscess, and disease of the hip-joint.

The *treatment* consists of free incision and subsequent drainage. When traceable to disease of the kidney, nephrectomy may be necessary.

HYDRO-NEPHROSIS.

Hydro-nephrosis results from non-infective obstruction of the ureter, either arising from some congenital defect, or from calculus, or from displacement of the kidney with kinking of the ureter, or from pressure upon the ureter from without by a pelvic tumour of some kind or by bands of lymph from old peritonitis. It is more likely to occur with an intermittent than a permanent obstruction. It is met with at all ages, and is more common in females than males. It is characterised by progressive dilatation of the duct above the obstruction, followed by compression and atrophy of the renal substance, with distension

of its fibrous capsule, resulting in the formation of a sacculated chamber, sometimes of enormous size, which is filled with fluid. The latter consists of altered urine, which is pale, watery, and of low specific gravity, not albuminous unless some pus or blood be present, with traces of urinary salts. The sac rarely bursts, and it either remains as a chronic cystic tumour, or the obstacle may be temporarily overcome, leading to discharge of the fluid, *per vias naturales*, and shrivelling up of the cyst. As a rule only one kidney is affected, and the other becomes hypertrophied.

The only sign of hydro-nephrosis is the formation of a smooth, painless, fluctuating tumour occupying the loin, and displacing the colon which lies in front of it. If large, it may be mistaken for an ovarian cyst. A sudden subsidence of the tumour with a copious discharge of somewhat altered urine, though rare, is highly characteristic.

If by reason of its size any *treatment* is required, nephrectomy may be desirable.

LARDACEOUS KIDNEY.

The conditions under which lardaceous kidney occurs are identical with those of lardaceous degeneration elsewhere. In its nature and origin, it has no connection with Bright's disease, though lardaceous changes are occasionally met with in the kidneys of such patients. The kidney is smooth, pale, increased in size and weight. On section, the cortex is thicker, and the Malpighian tufts in which the change begins appear as glistening points. At a later stage the renal substance as a whole becomes more or less uniformly waxy. The liver and spleen are almost always concurrently affected.

When the degeneration has sufficiently advanced, the urine is increased in quantity, pale, with a specific gravity

varying from 1005 to 1015. It contains little or no albumen at first, but in the later stages it may be very abundant. It is either clear, or yields a scanty deposit in which a few hyaline or granular casts may be found. Dropsy as a rule is not well marked, but it is commonly present, and may be extreme. Micturition is frequent. The complexion is pale and muddy; and diarrhœa is not infrequent from lardaceous implication of the bowel. The affection differs from Bright's disease in the absence of increased arterial tension and cardiac hypertrophy, the rarity of uræmic phenomena, and in its association with lardaceous changes in other organs—liver, spleen, and intestines, and a frequent history of prolonged suppuration. Death ultimately occurs from exhaustion, or some intercurrent complication. The treatment is that of the primary disease, coupled with the administration of tonics, of which a combination of iron and arsenic is the best.

NEW GROWTHS OF THE KIDNEY.

New growths in the kidney may be benign or malignant. The former, such as adenoma, fibroma, and lipoma, are of no clinical importance, and are only discovered *post mortem*. The chief malignant growths are sarcoma, carcinoma, and hypernephroma. The last has attracted much attention of late, and probably includes many growths that were formerly regarded as sarcomatous. It originates in aberrant nodules of adrenal tissue which are not infrequently found in the kidney. Sarcoma is almost always primary, and is most common in children. Carcinoma is relatively rare and occurs in later life. It may be primary or secondary. Hypernephroma may occur at any age, but is most common between 40 and 60.

The most suggestive indications of renal new growth are *pain*, *hæmaturia*, and *the presence of a local tumour*.

Pain may be altogether absent ; and, when present, varies greatly in different cases. In sarcoma and hypernephroma it is not a prominent feature, and is usually confined to aching across the loin, whereas in carcinoma it may be very severe. The passage of blood-clots along the ureter, however, may give rise to colicky pain of variable severity, in any case in which hæmaturia is abundant.

Hæmaturia, though not a constant feature, occurs in a majority of malignant tumours of the kidney. It is generally intermittent. The discovery of blood-casts of the ureter in the urine is very helpful in diagnosis.

A renal tumour occupies the flank, is generally rounded in shape, with a smooth surface, but it is sometimes irregular. As it grows, it extends forwards, pushing the colon in front of it, so that there is no resonance on percussion behind, between the tumour and the spine. Sarcoma and hypernephroma may form tumours of large size, but while the growth of the former is usually rapid, in the latter it is very slow. Carcinoma rarely gives rise to much enlargement.

Other symptoms may arise from pressure of the growth on adjacent parts, such as varicocele, obstruction of the bowel, dropsy, or jaundice. Marked emaciation and debility are usual features ; and there may possibly be evidence of malignant disease elsewhere. Death occurs from exhaustion. Secondary malignant growths in the kidney are bilateral, and give rise to no symptoms.

Nephrectomy may be performed in selected cases in an early stage, but the mortality of the operation is very high. Apart from this, only palliative remedies are available.

TUBERCULOSIS OF THE KIDNEY.

Tubercles may occur in the kidney as part of acute general tuberculosis ; but under these circumstances the

signs of renal disease are slight, and the symptoms merge into those of the general infection, so that a diagnosis during life is rarely possible.

On the other hand, a chronic local tuberculosis is not uncommon. It is due in all, or almost all cases to a secondary infection through the general circulation (*descending infection*), derived more often from some pre-existing tuberculous focus in the lymph-glands, lungs, or bone, though it may not be readily discoverable. Modern statistics show that in the early stages, the disease is unilateral in the very large majority of cases. Later on, the ureter, bladder, or the opposite kidney may become involved. Tubercles first appear at the apices of the pyramids and calices of the kidney. A pyelitis is quickly established; and, as the disease proceeds, the renal substance becomes gradually destroyed, and converted into a series of encysted caseous masses, which may soften, or undergo calcification. The ureter is liable to get plugged at times with tuberculous debris. The disease is most common between the ages of 20 and 40 years.

SYMPTOMS.—The onset is very insidious. For a long time symptoms may be entirely absent; and, when present, are at first more suggestive of bladder than renal trouble. The earliest indication is usually painful and too frequent micturition. The urine is always acid, apart from cystitis, and contains albumen—moderate at first, but gradually increasing as the kidney becomes more involved. It may be clear or cloudy. In all cases, provided the ureter remains patent, pus, blood, and renal epithelial cells may be found, if carefully looked for, and often in considerable quantity. Tubercle bacilli may also be found in a certain proportion of cases, if closely sought. When the disease is well established, granular detritus, cheesy particles, and shreds of tissue may be present; and, if so, are of great diagnostic value. Palpable enlargement of the

kidney is absent as a rule until a late stage, and then is generally quite moderate. More or less aching discomfort in the loin is frequent ; but actual pain is far less common, apart from colicky attacks, due to the passage along the ureter of blood-clot or débris. Constitutional disturbance with some fever gradually develop as the case proceeds, but the general health is often fairly maintained for a long time. If left to itself, the course of the disease is progressive and very chronic, though often marked by periods of decided improvement. Sooner or later, however, the bladder becomes affected or suppuration occurs around the kidney which hastens the end. The usual cause of death is exhaustion or septicæmia.

Early *diagnosis* is essential for successful treatment ; and rests upon very careful examination of the urine, supplemented when necessary by cystoscopic examination, ureteral catheterisation, tuberculin tests, and consideration of the personal and family history. The only method of *treatment* which offers success is by nephrectomy ; and the earlier it is performed, the better the chances of recovery.

RENAL CALCULUS.

Under certain conditions, one or other of the mineral constituents of the urine may be precipitated in the renal tubules, leading to necrosis of adjacent epithelial cells, with the products of which it becomes intimately associated, forming a particle or nucleus of more or less solid nature. The latter ultimately escapes into the urinary passages, in any part of which it is liable to be detained ; and as the result of progressive deposition of inorganic salts upon its surface, lead to the formation of a stone or calculus. Several nuclei may be formed concurrently ; and, if adjacent to each other, may unite and form the nucleus of a single

calculus on their escape from the kidney. On the other hand, they may remain separate, and give rise to multiple calculi. By far the most common stones which are formed in this way consist of uric acid salts and calcium oxalate in varying proportions, arranged for the most part in concentric layers, caused by variations in the composition of the deposit. Stones in which uric acid predominates are usually smooth and pebbly; while those consisting chiefly of oxalates often have an irregular surface (*mulberry calculus*). More rarely we meet with stones of calcium phosphate mixed with ammonio-magnesian phosphates, calcium carbonate, or cystine. For the most part calculi are small and rounded in shape; but not infrequently they are of large size, forming branched moulds of the renal pelvis. The latter generally result from phosphatic deposits upon a uric acid, or oxalate basis. Such deposits are of common occurrence whenever ammoniacal urine lies sufficiently long in association with organic matter.

Renal calculi are most common in men, from twenty to forty years of age. Though pyelitis, pyo- and hydro-nephrosis are not infrequent results of renal calculi, it is astonishing how long the latter may sometimes exist without obvious discomfort.

SYMPTOMS.—Renal calculus may occasion no symptoms of any kind, and even when symptoms indicative of calculus are limited to one kidney it is never safe to assume that the other kidney is necessarily free. The earliest symptom is generally darting or aching pain in one or other lumbar region, with a tendency to radiate into the groin and thigh of the same side. It is often associated with irritation at the end of the penis, frequent micturition, and the appearance of blood or pus cells in the urine. These symptoms recur from time to time in an intermittent form, and are apt to be induced by any sudden movement or effort which displaces the calculus. Severe attacks

may occur suddenly when the stone enters and passes along the ureter (*renal colic*), characterised by intense lumbar pains radiating in various directions, but chiefly downwards into the groin, testes, and inside of the thigh. It is also often associated with vomiting, perspirations, collapse, hæmaturia, and a frequent desire to pass water. Usually, after a few hours the attack ceases as suddenly as it began, by the arrival of the stone in the bladder, where it may either remain or be presently discharged by the urethra. If the attack lasts long, there may be some pyrexia. Less often the stone becomes impacted in the ureter, and in that case the attack lasts longer, subsides more slowly, and may be followed by pyelitis or pyonephrosis. When one kidney is congenitally defective, or extensively diseased, or the ureter blocked, the impaction of a calculus in the other may cause obstructive suppression of urine, and death from uræmia.

The *diagnosis* of renal colic is fairly easy. But, apart from colic, there may be difficulty. Pain and hæmaturia are the most important symptoms; but it is not safe to diagnose a calculus from pain alone, and the amount of blood may be so small that it can only be discovered by the microscope. In certain cases examination by the Röntgen rays may be very helpful.

TREATMENT.—During the paroxysms of renal colic, warm compresses or baths and the free administration of belladonna are mainly indicated. The tincture of belladonna should be given in full doses (say m 10–20 every hour), until its toxic properties begin to be manifested. If this fails, hypodermic injection of morphia is advised. During the intervals between the paroxysms, the diet should be moderate and simple. A diet limited to milk, eggs, cheese, and vegetables may sometimes be adopted with advantage. Acid fruits and rhubarb should be avoided.

Regular exercise in the fresh air is beneficial. If there be reason to think that the concretion is composed of uric acid, benefit may possibly be derived from the solvent effects of alkalinised urine. For this purpose free draughts of alkaline water such as Vichy or Contrexeville, or of plain water to which citrate of potash is added are desirable; but oxalate of lime concretions cannot be dissolved in this way. In chronic cases, unrelieved by medical treatment, the surgical measures of nephro-lithotomy or nephrectomy deserve careful consideration.

MOVABLE KIDNEY.

The term "movable kidney" or nephroptosis, is applied to a kidney the normal attachments of which have become relaxed. The degree of mobility varies from moderate displacement downwards, just permitting the lower end to be held, to such looseness as permits free movement within the abdomen (*floating kidney*). It is far more common in women than in men, and the right kidney is more often affected than the left. There is some obscurity as to the exact way in which the condition is brought about, and probably it is not always the same. The most important predisposing factors would seem to be general laxity of the tissues, special laxity of the abdominal wall (especially in relation to repeated pregnancies), nervous exhaustion, over-exertion, loss or deficiency of peri-renal fat, and compression of the upper abdomen by corsets or belts. It may be associated with displacements of other abdominal viscera. Hydro-nephrosis may develop in the displaced kidney, from kinking of the ureter.

SYMPTOMS.—These may be altogether absent; but when they occur they are mainly of a neuro-dyspeptic order, and not by any means in direct proportion to the degree of mobility. Many symptoms associated with movable

kidney depend upon underlying morbid conditions which are common to both, and should not be hastily regarded as directly attributable to the displacement as such. General debility, atonic dyspepsia, flatulence, constipation, headaches, irregular neuralgic pains, irritability of temper, undue fatigue, and palpitations probably belong to this class. Among the more characteristic symptoms is a sense of dragging in the abdomen with aching across the loin, which may pass on to acute pains radiating into the legs. Such pains are relieved by rest in the recumbent posture. From the reaction of persistent distress upon an unstable nervous system, a patient may pass into a condition of profound depression or melancholy. Again, we occasionally meet with severe agonising sudden attacks of pain, closely resembling renal colic. They are believed to be due to torsion, and consequent temporary obstruction of the ureter.

TREATMENT.—Corsets and tight clothing should be rigidly forbidden; a carefully adjusted abdominal support should be worn; and massage and exercises should be practised to strengthen the abdominal walls. In severe cases, the operation of fixing the kidney by stitches may be of great service.

CHYLURIA.

Chyluria is a rare affection characterised by the discharge of milky urine, which on standing sometimes coagulates into a tremulous jelly, an event which may occur before leaving the bladder. The urine contains a large quantity of molecular fat which is readily soluble in and separable by ether, with fibrin, albumen, and often a little blood. There are no casts.

It is due to some direct communication between the lymphatic and urinary systems, which in some cases is

caused by a small nematoid worm (*Filaria sanguinis hominis*).

No form of treatment is attended with much benefit, but the disorder is not inconsistent with fair health for a long period.

HÆMOGLOBINURIA.

This condition is characterised by the discharge of dark, porter-coloured urine, due to blood-pigment, depositing an abundant sediment of dark granular matter, which consists mainly of granular pigment and urates *without* red blood-cells. Two types of cases are met with. In the one—*symptomatic*—it is due to some toxic or infectious condition, such as poisoning by potassium chlorate, yellow fever, or hæmorrhagic types of eruptive fevers. In the other—*paroxysmal*—it is characterised by the occurrence at irregular intervals of paroxysmal attacks, ushered in abruptly with chills and fever. In some cases, the temperature is *lower* than normal, with a subjective sensation of cold, general malaise, yawning, and nausea. This is shortly followed by the passage of urine as above described. After a few hours, or somewhat longer, the symptoms abate, the urine becomes clear again, but albuminuria may persist for a time. Such attacks may recur over a prolonged period, but are rarely attended with marked deterioration of the general health. They may be brought on by exposure to cold, severe exertion, or mental anxiety, and are most common in young male adults. The pathology is obscure.

As to *treatment*—during the paroxysm the patient should be placed in bed and kept warm, and at all times he should be warmly clad, and carefully avoid any exposure to draughts. Medicinal treatment appears to be of very little service.

SUPPRESSION OF URINE. ANURIA.

Suppression of urine may depend on a direct suspension of the secretory functions of the kidney (*non-obstructive suppression*) ; or on the other hand it may be due to some mechanical obstacle to the outflow of urine (*obstructive suppression*).

Non-obstructive suppression of urine may occur as the result of advanced organic disease of the kidneys, acute nephritis, or of intense congestion set up by the administration of turpentine or cantharides. Sometimes it occurs independently of structural change in the kidneys as the result—for instance—of nervous shock on passing a catheter. Unless it speedily subsides, it gives rise to symptoms of acute uræmia, and death occurs in the course of a few days. Treatment by hot applications or dry cupping over the loins, diaphoretic measures, and free purging is indicated. In hysteria one occasionally meets with temporary suppression of urine, but it is not followed by uræmic trouble for some obscure reason. The condition should be carefully distinguished from hysterical retention of urine.

Obstructive suppression of urine generally arises from impaction of a stone in one ureter, when the other has previously become blocked, or when the opposite kidney is disorganised by disease or congenitally deficient. Cancer of the bladder may sometimes block up the lower orifices of both ureters. If any urine at all passes, it is pale, of low sp. gr., and contains little urea. No symptoms of any importance occur during the first week, but about this time trembling and muscular weakness, contraction of the pupils, slow laboured respiration, dryness of the mouth, and complete anorexia supervene. Coma and convulsions do not occur. Death usually takes place

in two or three days after the appearance of these symptoms. Apart from the possible relief of the obstruction by surgical operation, nothing can be done.

INCONTINENCE OF URINE.

Three varieties of incontinence should be recognised.

1. *Simple incontinence.* This results from relaxation of the sphincter of the bladder, owing to some weakness or destructive lesion of the automatic nervous centre in the lumbar enlargement of the spinal cord, by which, under normal circumstances, its tone is maintained. This is a common occurrence in certain diseases of the spinal cord, such as myelitis. 2. *Intermittent incontinence*—in which, owing to defective cerebral control, or to undue irritability of the vesical reflex mechanism, or to both conditions combined, the bladder is automatically emptied with increased frequency. When it occurs during sleep, and also in certain delirious or comatose states, the patient is unconscious of the act. 3. *Overflow incontinence*—in which the bladder becomes over-distended, either from voluntary retention, mechanical impediment, or some sensory defect in the vesical nervous mechanism, so that the detrusor becomes temporarily paralysed. Under these circumstances the urine accumulates, till the resistance of the sphincter is overcome, when it dribbles away.

Nocturnal Incontinence.—Enuresis.—This affection almost exclusively occurs in children before puberty, and rarely persists later than this. The trouble may occur only at night, or it may be associated with frequent and imperfectly controlled micturition during the day as well. It is essentially a form of intermittent incontinence. All cases should be studied from two points of view:—first as regards the general health, and especially with regard to the state of the nervous and digestive functions;

secondly, with regard to the condition of the urine, and the presence or absence of local sources of irritation, such as phimosis, vesical calculus, or thread-worms.

As to *treatment*, the first thing is to remove any existing source of local irritation. Then the diet should be carefully regulated, sugar and carbohydrate food should be restricted, late meals should be forbidden, and no fluid should be taken after tea-time. Fruit—especially uncooked fruit—should be avoided. The child should lie on a mattress and be only lightly covered. Medicinally, the most useful drug is belladonna, beginning with $\mathfrak{m}\mathfrak{v}$. of the tincture three times a day. This dose should be steadily increased until either the enuresis ceases, or the limits of tolerance have been reached. With favourable results, the dose reached should be maintained for a week or two, and then gradually diminished. In some highly nervous children, especially those with a history of rheumatism, a course of salicylate of soda with potassium bromide and antipyrin may be effective. Among other drugs which are worthy of trial in obstinate cases (for there are no trustworthy indications available) are tincture of nux vomica ($\mathfrak{m}\mathfrak{ij}$., steadily increased), fluid extract of ergot $\mathfrak{m}\mathfrak{xx}$., and fluid extract of rhus aromatica $\mathfrak{m}\mathfrak{x}$. These doses are for a child of five or six years of age, and should be repeated three times a day. Many children greatly improve with change of air, and the adoption of a tonic regime. The child should pass water just before going to bed, and be taken up again two hours later, and (if possible) once in the course of the night.

SECTION X.

DISEASES OF THE NERVOUS SYSTEM.

INTRODUCTION.

1. GENERAL STRUCTURE AND FUNCTIONS.

FOR full details the reader must refer to special text-books, but a brief consideration of certain points is indispensable even for an elementary study of nervous diseases.

The primary units of which the nervous system is composed are called *neurons*. A typical neuron consists of a protoplasmic cell, apparently destitute of cell-wall, possessing a nucleus and nucleolus, and exhibiting a variable number of filamentary processes. Of these processes, one is distinguished from the rest by its much greater length, uniformity of diameter, and scanty branching (*axon*). An axon gives off a few branches, usually at right angles, which are called *collaterals*; and both the main thread and collaterals finally break up into brushes of fibrils (*arborisations*). At a variable distance from their origin the axons become invested with medullary matter and a fibrous sheath, and constitute the fibres which form the white substance of the brain, cord, and peripheral nerves. The remaining processes are relatively short, and break up at once into terminal brushes (*dendrons*). Nerve-cells with their dendrons are collected into compacted groups, and form the grey substance of the brain and cord. Individual neurons are brought into relation with each

other by interlacement of the terminal axon-fibrils of one neuron with the dendritic fibrils of another. The relationship is one of contact only, without actual continuity. Modifications of this arrangement occur in certain parts. Thus the cells of the ganglia on the posterior spinal roots have only two processes united to the cell-body by a single short stalk. One of them, representing the axon, passes into the cord, and enters into relation with the dendrons of cornual cells; while the other, representing the dendron, passes down the peripheral nerve, and enters into relation with sensory end-organs. Physiologically, the dendrons are receptive or afferent, and conduct impressions to the cell-body; while axons are emissive or efferent, and conduct impulses from the cell-body.

From the point of view of its development, the cerebro-spinal nervous system broadly consists of an axial tube of grey matter, clothed with a sheet of white fibres, which is again covered, in its anterior or cerebral portion, by a cortical layer of grey matter. Corresponding with this elementary conception of nervous structure, there are, in general terms, three groups of neurons:—(1) Those connecting cortical with axial grey matter; (2) those connecting axial grey matter with peripheral end-organs; and (3) neurons of association, connecting different parts of cortex or axis with each other. Any nervous communication between cortex and periphery, whether efferent or afferent, must therefore of necessity pass through at least two neurons, and all but the simplest nervous phenomena involve the co-operation of neurons of association as well.

In the grey matter of the cerebral convolutions we find the neuron centres for volitional movement, conscious sensation, and intellectual activity. The ascending frontal convolution, lying immediately in front of the fissure of Rolando, constitutes the motor area. Irritative lesions within this area are attended with definite *combined move-*

ments of some kind (*not* contractions of individual muscles), whilst destructive lesions of corresponding centres are followed by paralysis of similar movements. The upper third of the area is concerned with the movements of the leg and trunk, the middle third with those of the arm, and the lower third with those of the face, tongue, and larynx. It is from this area that the motor or pyramidal tract takes its origin. The frontal cortex immediately in front of the motor area is probably concerned with psycho-motor processes, which have for their object the organisation of complex and highly differentiated movements. Thus, the hinder end of the third frontal convolution controls the motor mechanism of speech, and the corresponding part of the second convolution controls the mechanism of writing. The convolutions posterior to the motor area mainly subserve conscious sensation—general sensation, in the posterior central and adjacent parietal convolutions; vision, in the gyrus cuneus of the occipital lobe; hearing, in the upper temporo-sphenoidal convolution. Yet again there are sensory centres on a higher physiological plane. Thus in the left angular gyrus and adjacent parts we find the centres for the intellectual interpretation of visual impressions, and similarly the interpretation of auditory impressions is assigned to a special part of the left temporo-sphenoidal convolution. Of the localisation of the higher volitional and ideational centres, we have no definite knowledge.

The white substance underlying the convolutions is made up of axon-fibres passing from the cortex in various directions, but for the most part they converge towards the base of the brain. Those from the motor area deserve especial attention, as they form the path along which motor impulses travel downwards from the brain to the muscles. Starting from the ascending frontal convolution these motor strands converge to the band of white matter

intervening between the optic thalamus and the corpus striatum (*internal capsule*), forming, however, only the knee, and anterior two-thirds of the posterior division of this band. Thence they pass through the *crusta* of the *crus cerebri*, and on arriving at the medulla form the anterior pyramids which mainly cross to the opposite side in the lower part of the medulla to form the crossed pyramidal tract in the lateral column of the spinal cord, and eventually end in the arborisations surrounding the motor cells in the anterior horn of the cord. From the latter cells the axons of the lower motor neurons take their origin, issuing as the anterior roots of the spinal nerves. The pyramidal fibres which do not decussate in the medulla pass down on the inner side of the anterior column of the cord on the same side, but their ultimate distribution is somewhat uncertain. The course thus traced constitutes what is called the "motor path."

With regard to the "sensory path," we know much less. In the upward passage of sensory impressions, at least three tiers of neurons are engaged. The lowest of these have their cell-bodies in the ganglia of the posterior roots of the spinal nerves, the dendritic processes of which form the afferent fibres of the spinal nerves, while some of their axons pass directly into the grey matter of the cord, and others extend upwards in the posterior white columns on the same side to the gracile and cuneate nuclei in the medulla. Of the middle series of neurons, some arising in the grey matter of the cord send their axons up to the brain in the antero- and postero-lateral tracts; while others arising in the grey matter of the gracile and cuneate nuclei form the fillet, which immediately decussates, and passes upwards to end in the optic thalamus of the opposite side. Among the highest series of neurons are those which, arising in the optic thalamus, connect it with the cortex of the brain of the same side, passing through the

internal capsule in its posterior third, immediately behind the motor fibres, and somewhat in front of the optic radiation.

At the base of the brain we find a series of ganglionic masses of grey matter, some of which are of large size, such as the corpora striata, optic thalami, and the corpora quadrigemina; and passing down thence to the termination of the cord below are several smaller ganglia closely succeeding and connected with each other. Each of these centres is concerned with the reception of different sorts of afferent impressions, or is capable of generating some kind of efferent impulse. The corpora striata have no direct connection either with the motor tract or with the cerebral convolutions, but on the other hand they have an extensive connection with the cerebellum of the opposite side. The optic thalami receive fibres from the cerebellum, the sensory tract, and perhaps from the optic nerves; and from them fibres pass in all directions to the convolutions.

The *sympathetic system* is so closely related to the cerebro-spinal system, both from an anatomical and physiological point of view, that it is difficult to consider it separately. While we have in the sympathetic ganglia, centres which preside over the organic functions of the parts with which they are in connection, it would seem that in the medulla oblongata there is a supreme controlling centre for the whole sympathetic system operating through the communicating branches of the latter with the spinal nerves. The most important function of this system is vaso-motor, and is due to its relation to unstriped muscular tissue, which contracts on stimulation of the sympathetic nerves by which it is supplied, while section of the latter is followed by its relaxation. In this way the calibre of the blood-vessels, and therefore the amount of blood supplied to various parts, may be regulated. Apart from the *indirect* influence thus brought to bear upon tissue

nutrition by the sympathetic nervous system, there is reason to think that it may also exert a *direct* or *trophic* influence on the functional activity of the cells in secreting glands and elsewhere.

DEGENERATIONS. TROPHONEUROSES.

1. *Nervous System*.—The cell-bodies of neurons are liable to degeneration from various causes, such as exhaustion, malnutrition, inflammation, mechanical injury or pressure, and toxic troubles. This is followed by secondary degeneration of their processes, whether axons or dendrons. Moreover, any lesion which breaks the continuity of a neuron process with its cell-body invariably leads to degeneration of the distal segment extending to the special end-organ—motor or sensory—with which it is associated. Neuronic degeneration leads to overgrowth of neuroglia in the affected area, which afterwards shrinks and becomes firm, giving rise to the condition known as *sclerosis*.

2. *Bones and Joints*.—In certain nervous diseases, such as tabes dorsalis and syringomyelia, we meet with atrophy and undue fragility of the bones, or with atrophy of the joints, attended with rapid wearing away of the articular surfaces, and consequent dislocations. The latter may be accompanied with hyperplastic new formations around the joint, producing a condition closely resembling rheumatoid arthritis. These changes are probably due to disease of some part of a reflex nervous arc by which the nutrition of the joint is regulated. The hyperplastic overgrowths appear to be of secondary origin. A good example of nervous arthropathy is afforded by the so-called “Charcot’s joint” in tabes dorsalis.

3. *Skin*.—Affections of the lower sensory neurons often give rise to changes in the skin. Thus, under the influence of irritative or inflammatory lesions, the skin may become

atrophied and shiny (*glossy skin*), or herpetic eruptions may occur. When cutaneous areas are cut off from their normal innervation they become very liable to serious damage from slight causes such as pressure—examples of which are afforded by acute sacral bedsores in paraplegia, and perforating ulcer of the foot in *tabes dorsalis*. In other cases (of uncertain pathology) we may meet with pigmentary anomalies, *ichthyosis*, or changes in the hair and nails.

DISTURBANCES OF SENSATION.

Hyperæsthesia signifies an exalted susceptibility of the nerve-centres concerned with common or special sensation, by reason of which sense-impressions are abnormally acute or painful. When the skin is thus affected, the slightest touch causes pain, and similarly the eye may be unable to bear bright light, or the ear loud sounds without much distress. It is of common occurrence in hysteria, and in certain inflammatory and other affections of the brain and cord. It will be seen that the term *hyperæsthesia*, as usually (but not quite correctly) employed, means something quite different from increased delicacy of sensation.

Dysæsthesia or perverted sensibility includes such sensations as formication, numbness and itching; and singing noises in the ears (*tinnitus*), sparks of light in the eyes, etc.

Anæsthesia denotes an impairment or loss of sensibility, which may be either superficial, deep-seated, or involve the whole thickness of a part. Its degree and extent vary greatly. It is often attended with numbness, tingling, or pricking sensations. Disturbance of sensibility to variations in temperature and of muscular sense do not necessarily accompany loss of tactile sensibility. *Anæsthesia* may arise from organic disease of the brain, cord, or nerves; or from functional nervous disease such as hysteria.

Hemi-anæsthesia signifies a loss of sensibility over one side of the body. In its complete form (including the loss on one side not only of *common* but also of *special* sensation) it is always of functional origin, and is associated with the hysterical condition. It is often associated with areas of tenderness on pressure over the ovary or breast. As affecting common sensation only, hemi-anæsthesia sometimes occurs in lesions of the brain, affecting the posterior third of the internal capsule, or the parts in direct connection with it. In such cases, it is almost always associated with some degree of hemiplegia.

Muscular sense is that sense by which we tell the position of our limbs without the aid of vision; and by which we are enabled to judge of the force, extent, rapidity, and duration of muscular effort required for any particular purpose, such as the estimation of weight, for instance. The former may be tested by performance of movements when blindfold; and the latter, by suspending bags of shot of variable weight from the hands or feet. *Loss of muscular sense* is one of the most characteristic symptoms of *tabes dorsalis*.

Pain.—Pain is a purely subjective symptom, and accordingly one can only be guided by the temperament, expression, and posture of a patient in forming an idea of its amount or quality. When pain is elicited by pressure it is spoken of as *tenderness*. Increased sensitiveness to pain is sometimes described as *hyperalgesia*, whereas insensibility to pain is distinguished by the term *analgesia*. The distribution of pain is obviously an important guide to diagnosis: but since it is often referred to a point at considerable distance from the irritation which gives rise to it, care is required in drawing conclusions. Each internal organ has a definite nervous connection with an area on the surface of the body, to which pain is referred on irritation of the viscera with which they correspond.

Many of these have been already attended to in the course of this work.

CONVULSION—MUSCULAR SPASM.

Under this head are included all involuntary contractions of the skeletal muscles. Such contractions are termed *clonic* when they are intermittent, and *tonic* when persistent.

1. *Tremor* is the mildest form of clonic muscular spasm, and consists of a series of rhythmical muscular contractions, which vary widely in range of movement. Tremor is sometimes confined to individual bundles of muscle-fibres, being unattended with actual change in position of the part, and occurring independently of rest or exertion (*fibrillary tremor*). It is generally associated with fatigue or central lesions of lower motor neurons. Fine tremor, more marked on exertion or excitement, is a common feature of Graves' disease, toxic poisoning by alcohol, tobacco, lead, or mercury, and general paralysis. In paralysis agitans we also meet with a coarser rhythmical tremor which may take the form of grouped movements, such as rolling the thumb on the fore-finger, pronation and supination, and the like. It continues during repose, but ceases in sleep, and is temporarily arrested by voluntary movements. Somewhat analogous coarse tremors are sometimes observed in hysteria. The tremor of disseminated sclerosis is peculiar in that it only occurs on attempted voluntary movement (*intention tremor*), and becoming more marked as the movement is continued. When this form of tremor affects the ocular muscles, it is termed *nystagmus*. ¶

The pathology of tremor is obscure, but it is probably the result of impaired vitality of nervous structures, owing to which, nervous impulses are no longer generated or conducted with sufficient rapidity to fuse with each other and produce a continuous muscular contraction.

2. *Choreic movements* are of a disorderly nature, but differ from ordinary convulsive contractions in their tendency to conform to a more or less physiological grouping, and in chiefly affecting the movements of expression and gesture. They vary in degree from simple twitching to violent contortions. They cease during sleep, and are aggravated by attempts at voluntary movement. In some cases they are limited to the face, to a single limb, or to one-half of the body.

3. *Athetosis* is a term applied to peculiar, slow, writhing movements of the fingers or toes. It is increased by emotion, and often persists during sleep. It is usually a sequel of cerebral paralysis in early childhood, and appears to be associated with lesions of the thalamus.

4. *Convulsions* consist of irregular clonic muscular contractions attended with corresponding movements of the affected part. They are due to direct excitement or irritation of the cortical motor centres concerned, as the result of local organic disease, such as tumour, or of certain toxic states of the blood as in uræmia, or of functional disturbance as in epilepsy and hysteria. Convulsions present every variety of distribution, and may involve the whole body (*general convulsions*). *Eclampsia* is an old-fashioned term sometimes applied to general convulsions which are obviously of secondary origin, such for instance as convulsion occurring in connection with puerperal conditions.

5. *Tonic spasm* is a steady persistent involuntary muscular contraction which varies from simple stiffness to extreme rigidity. The simplest form of tonic spasm is exhibited in "*cramp*." Cramp is a common symptom of visceral irritation within the abdomen: and, in a more severe form, constitutes the characteristic feature of tetanus and strychnia-poisoning. When a group of muscles is paralysed, their antagonists are apt to become

permanently contracted. Another important variety of tonic spasm occurs in association with degeneration of upper motor neurons is known as "*spasticity*." Tonic muscular contraction is also a prominent feature of torticollis, writer's cramp, and certain cases of hysteria.

PARALYSIS.

Paralysis denotes loss of motor power. As applied to voluntary muscles it implies inability on the part of the will to determine movement. The term *paresis* is often applied to imperfect or partial paralysis. Paralysis may be due to functional disorder, to organic disease of the brain, cord, or peripheral nerves.

Paralysis may be classified according to its dependence on interference with upper or with lower motor neurons. In the former it is due to trouble either in the motor area of the cerebral cortex or in some part of the motor tract which issues from it (*supra-nuclear paralysis*), and is a paralysis of *movement*, in which various muscles are functionally associated. In the latter, it is due to lesion of motor nerves or of the nuclei in which they take their origin (*infra-nuclear* or *nuclear paralysis*), and the paralysis is confined to the *muscles* supplied by the nerve or nerves affected.

Upper motor neuron, or *supra-nuclear paralysis* resulting from injury or disease of the motor cerebral cortex is usually confined to one limb, or to some special movement, according to the cortical centres which are involved. Supra-nuclear paralysis is more commonly the result of lesions of the motor tract below the cortex, especially in the region of the internal capsule, arising from rupture, thrombosis, or embolism of some branch of the middle cerebral artery. Under these circumstances, paralysis is limited to one side of the body, opposite to that on which the lesion occurs (*hemiplegia*).

All the movements of the affected side are not equally involved. In a typical case, while the arm, leg, and lower part of the face are more or less completely paralysed, bilateral movements such as frowning or wrinkling of the forehead, closing the eyes, and the respiratory movements of the trunk are approximately exempt. This is believed to be due to the intimacy with which the centres for bilateral movements are associated, on the two sides of the brain. That these centres on the affected side do not escape altogether is shown by the fact that when called into action unilaterally on the paralysed side, movement is diminished or lost. Thus, while both eyes can be closed *simultaneously* the eye on the affected side cannot be closed *independently* of the other, as in winking; and again, though bilateral respiratory movements are scarcely affected there is obvious weakness of the same muscles on attempting to bend the trunk to the paralysed side. Differences also in the degree of paralysis, and the order of its recovery depend in some measure upon the extent to which the movements of the part are differentiated. Thus paralysis is less complete and recovers sooner in the less differentiated movements of the leg than in the more highly differentiated movements of the arm; and in the movements of the shoulder than in those of the hand or fingers, for the same reason. The angle of the mouth is drawn upwards and to the sound side, the cheek hangs loosely, the lower lines of expression are ill-marked, there is difficulty in pursing the lips, and the tongue when protruded is deflected to the paralysed side. Owing to the weakness of the tongue and lips, utterance is often defective (*dysarthria*); but this must not be confounded with aphasia, which so generally accompanies right hemiplegia.

Hemiplegia from lesions of the motor tract *below* the internal capsule is characterised by frequent association

with nuclear paralysis of motor nerves which take their origin in the neighbourhood of the lesion. Hence it is that in unilateral damage of the *crus cerebri* or *pons*, a crossed paralysis occurs, in which there is nuclear paralysis of one or more cranial nerves on the *same side* as the lesion, with supra-nuclear paralysis of the face, arm, and leg on the *opposite side*. Where the motor tracts approach each other in the medulla, a lesion may cause paralysis of *both sides* of the body; and if limited to a single motor tract in the spinal cord, the paralysis will be on the *same side* as the lesion. Hemiplegia is an occasional result of functional disturbance of upper motor neurons, more often in connection with hysteria, as will be described later on.

Lesions in the spinal cord are more often bilateral, in which case, all muscles supplied by nerves issuing below the lesion will be paralysed. This condition is termed *paraplegia*. When the whole thickness of the cord is involved, loss of sensation accompanies the loss of motion; and, should the lesion include the centres of micturition and defæcation in the lumbar enlargement of the cord, paraplegia is associated with incontinence of urine and fæces.

CONDITION OF PARALYSED MUSCLES.

Tone.—In supra-nuclear lesions of sudden onset the muscles of the paralysed limbs may be limp and flaccid for a short time. Otherwise lesions of the pyramidal tract are invariably attended with more or less persistent rigidity, or spasticity. Rigidity may also occur as the result of direct irritation of upper motor neurons, as in cerebral meningitis, or hæmorrhage into the ventricles of the brain. In nuclear or infra-nuclear paralysis the affected muscles at once lose their tone and become flaccid.

Electrical Reaction.—This may be ascertained in two

ways :—(1) By *Faradisation*, in which the currents are of momentary duration and of high tension ; or (2) by *Galvanism*, in which they are slowly interrupted and of comparatively low tension. In supra-nuclear paralysis, the electrical reactions remain unaffected for a long time ; but when the affected muscles become atrophied from disuse, electric contractility suffers in direct proportion to the amount of atrophy.

In nuclear or infra-nuclear paralysis the muscles quickly atrophy, and their electrical behaviour is characteristically disturbed. During the first week the reaction of affected muscles to faradism is rapidly diminished, and by the end of the second week the reaction is lost, nor is it recovered until regeneration of nerve takes place. With regard to galvanism the reaction of affected muscles is increased for a variable time ; but, as the muscles waste, the response slowly diminishes, though it may be some months before it is finally lost. Moreover, the normal sharp response is replaced by one which is slow and wavy ; and when minimal currents are employed, it will be noted that the anodal closing contraction is equal to or greater than the kathodal ; or as it is often written $ACC =$ or $> KCC$. The latter is known as the *polar change*, and is exactly the reverse of the normal order. The above phenomena, taken as a whole, make up what is called the *reaction of degeneration*.

REFLEX PHENOMENA.

Reflex phenomena are dependent upon the integrity of reflex arcs, each consisting of an afferent nerve and an efferent nerve brought into relation with each other by centrally situated nerve-cells.

Since the spinal cord forms, with the afferent and efferent nerves attached to it, a superimposed series of reflex arcs, it follows that where disease of the nerves can be excluded

observation of the behaviour of such reflexes will yield valuable information as to the condition of the cord segments with which they are in connection. These reflexes may be divided into two groups—*superficial* and *deep*.

The most important *superficial* reflexes are the *plantar reflex*—movements obtained by drawing the finger-nail along the sole of the foot, chiefly involving the first sacral nerves; *cremasteric reflex*—withdrawal of the testicle on stimulation of the inner side of the thigh—1st to 3rd lumbar nerves; *abdominal reflex*—by irritation of the abdomen at the outer edge of the rectus muscle—8th dorsal to 1st lumbar nerves; *epigastric reflex*—by scratching the skin over the lower rib cartilages—4th to 7th dorsal nerves; and the *corneal reflex*—blinking of the eyelids by gently blowing on the cornea—5th cranial nerve. The plantar reflex demands a few more words. Under ordinary circumstances it is attended by flexion of the big toe (*flexor response*) together with other movements of less significance. In the presence of a lesion of the corresponding pyramidal tract, the first involuntary response is an extensor movement of the big toe (*extensor response*). A similar extensor response occurs normally in infants, as the result of incomplete development of the pyramidal tract. The plantar reflex is abolished in nuclear or infra-nuclear paralysis affecting the legs, and often in hysteria. It may be difficult to obtain it unless the foot is dry and warm. All superficial reflexes are of *directly* reflex nature; their presence implies the structural and functional integrity of the arcs involved; their absence always implies some interruption of the corresponding arcs by peripheral or central disorder or disease; and their exaggeration generally points to diminished cerebral control.

The most important *deep reflexes* are the *knee-jerk*, and

ankle-clonus. They are *not* directly reflex in nature, but are short muscular contractions produced by excitation of the muscles themselves under the influence of sudden tension. This muscular response is in direct proportion to the muscular tonus, which in turn depends upon the maintenance of the reflex arc in a normal state.

The *knee-jerk* is elicited in health by smartly striking the patellar tendon with some blunt object, while the leg hangs loosely from the knee. The reflex arc which presides over it joins the cord at the level of the third lumbar nerve. If this reflex arc be interrupted at any point, by disease of the cord or peripheral nerves, the phenomenon is absent, as in locomotor ataxy and peripheral neuritis. It is also sometimes temporarily abolished by brain-shock, as in apoplexy. On the other hand, when the arc is removed from the inhibitory influence of the brain—as, for instance, in degeneration of the pyramidal tract in the cord, it is exaggerated. Increased knee-jerk is also common in neurasthenia and other functional neuroses. Similar jerks can be obtained by suitable stimuli in the arm (triceps jerk and supinator jerk) and jaw, which are increased or abolished under the same conditions as those affecting the knee-jerks.

Ankle-clonus is characterised by a persistent series of rapid rhythmical movements of the foot at the ankle-joint, from sudden and forcible passive flexion of the latter on the leg. It cannot be obtained in health, but under similar conditions to those in which the knee-jerk is exaggerated, it may be elicited. The primary source of this reflex is a stimulus afforded by sudden tension of the muscles of the calf. The reflex centre is situated in the cord at level of the first and second sacral nerves.

APHASIA.

The subject of aphasia is very difficult, and there has

been much controversy and considerable difference of opinion with regard to it. Only an elementary presentation of the matter will be attempted in the remarks which follow.

The faculty of speech is of twofold nature. On its inward or subjective side, it is a *perceptive* faculty, concerned with the understanding of spoken and written words in relation to thought. On its outward or objective side, it is an *expressive* faculty, concerned with the co-ordinated acts of speaking and writing. Failure on either side constitutes what is known as *aphasia*. Expressive failure is called *motor* aphasia; and perceptive failure is called *sensory* aphasia; while if both are involved together there is *total* or *complete* aphasia. Aphasia should be distinguished from mental incapacity or absence of ideas, and also from local defects in the apparatus of articulation (*dysarthria*).

The mechanism of speech consists primarily of three groups of cortical nerve-centres in which, under the influence of education, three different kinds of memories are acquired and stored up:—(a) a centre for the sounds of spoken words (*auditory memories*), seated in the left upper temporo-sphenoidal convolution; (b) a centre for visual images of written words (*visual memories*), seated in the left angular gyrus; and (c) a centre for memories of the movements required in articulate speech (*motor memories*), seated at the hinder end of the third left frontal convolution (Broca's area). With regard to the movements concerned with writing, while some say that it is controlled from the same centre as articulate speech, others would allot them to a separate but adjoining centre. The above centres are intimately connected with each other, and form a zone of speech-centres extending across the left brain, functionally receptive at its hinder end and emissive at its front end. From the expressive

portion efferent fibres pass down to the motor tract connecting it directly or indirectly with the primary centres for the movements of the tongue, lips, larynx, and hand; and the receptive portion receives afferent fibres from the centres of hearing and vision. All parts of the speech-zone are also connected by commissural fibres with corresponding parts on the opposite side of the brain. Lastly it should be borne in mind that the whole zone receives its blood-supply from one and the same vessel, namely, the middle cerebral artery.

Motor aphasia is very commonly associated with right hemiplegia, as the result of rupture, thrombosis, or embolism of the same branch of the middle cerebral artery. When it is due to lesion of the motor speech area itself, it is said to be *cortical*. When it is due to a lesion in the pathway from the speech-centre to the motor tract, it is said to be *sub-cortical*. In the *cortical* type, the mind is relatively clear: the patient understands what is said to him, and apparently what he reads, though often imperfectly in each case; but he is either quite unable to express himself by speech or writing, or does so incorrectly, and with difficulty. Automatic words and phrases which involve no intellectual process may be readily expressed, and often replace the word which is really wanted. Thus a reply to any question may invariably be limited to "Yes" or "No." The patient is generally quite aware of the mistakes he makes, but is unable to avoid them. In the *sub-cortical* type, the symptoms are the same, with two exceptions: the patient is able to write perfectly (if his hand is not paralysed), and, if sufficiently intelligent, may be able to show that he has a clear mental picture of the word he wants by means of some action, such as tapping the table as many times as there are syllables in the word.

Sensory aphasia is due to cortical or sub-cortical lesions

of the posterior or receptive portion of the speech-zone. It may be predominantly *auditory* or *visual*, but pure forms are relatively infrequent, especially of the auditory type. It is seldom associated with hemiplegia, unless the lesion is very extensive. When a patient fails to understand *spoken* words, though he hears them clearly, he is said to be *word-deaf*. In a complete form he would be speechless, and unable to repeat words or to write from dictation, though he may be able to copy. Spontaneous writing may also be lost. More often, auditory aphasia is incomplete; and, under these circumstances, and in the earlier stages of a progressive lesion, there may be constant chattering of an unintelligible kind, and if able to write, the writing is disordered and meaningless. When a patient fails to understand *written* or *printed* words, though he sees them clearly, he is said to be *word-blind*. Word-blindness is more common than word-deafness. When complete there is total inability to read, write, or copy; but if incomplete, individual letters or easy and familiar words may be recognised, and perhaps imperfectly written. Spontaneous speech is unaltered.

Sub-cortical forms of auditory and visual aphasia are also described, in which the cortical speech-centres are not directly involved, but are cut off from the primary centres of hearing and sight respectively. Spontaneous speech and writing are preserved; but in the former (auditory) the patient cannot understand spoken words or write from dictation; and in the latter (visual) though he can write, he cannot read what is written.

It must be understood that the above claims to be no more than an elementary exposition of the chief types in aphasia. In actual practice, we sometimes meet with extremely complex combinations of speech-defects, the study of which demands much more detail than can be given here.

HEADACHE.

Headache is not only one of the commonest, but often one of the most distressing symptoms of cerebral disorder. The pain is variously described as dull, heavy, shooting, throbbing, constricting, etc. It may be either more or less widely diffused, or localised to a particular spot, or confined to the course of some sensory nerve. As to its seat it may be general, frontal, vertical, occipital, or one-sided.

Headaches may be roughly divided into two groups:—(1) *cranial headaches*, due to some affections of the cranial muscles, nerves, bones, or periosteum; and (2) *cerebral headaches*, due to irritation of the brain itself. The chief causes of cerebral headaches are:—(a) disturbance of circulation (anæmia and congestion); (b) exhaustion or neurasthenia; (c) digestive derangements; (d) toxæmia (gout, uræmia, alcohol, tobacco, and constipation); (e) inflammation and organic disease; and (f) reflex irritation (dental caries, enlarged tonsils, post-nasal growths, eye-strain, and ovario-uterine troubles).

In practice it is often a matter of great difficulty to refer a headache to its precise cause. Sharp pains in the frontal or occipital region, increased by wrinkling the scalp, accompanied with tenderness over the muscles, varying with the weather, and associated with evidences of rheumatism elsewhere, are suggestive of myalgia. In neuralgia and migraine the pain is paroxysmal, one-sided, corresponds to the distribution of one or more nerve-trunks, with points of localised tenderness. Similar characters also attach to many reflex headaches. Anæmic headaches are most common in women, are usually persistent, relieved as a rule by the recumbent posture, are generally vertical, and accompanied with weakness, depression, attacks of dizziness, tinnitus aurium, and specks in the field of vision. Sleeplessness is common, though the patient is often very

drowsy during the day. Congestive headaches, on the other hand, are throbbing, extend over the whole head and are increased by stooping, lying down, exercise, or excitement of any kind. Neurasthenic headaches are often characterised by a sense of vertical pressure, with weariness and aching in the back of the neck. Headache from gastro-hepatic disorder is generally frontal and of a dull heavy character.

Headache from structural diseases of the brain is usually severe, persistent (though sometimes paroxysmal), aggravated by the recumbent posture, keeps the patient awake at night, and is often associated with convulsive or paralytic symptoms. Sometimes valuable information may be gained by the use of the ophthalmoscope, showing the presence of inflammatory changes in the optic disc or retina. But the conditions under which headache occurs are so complex and varied, that a diagnosis can only be arrived at after a careful study of associated conditions, and of the mode of evolution.

DELIRIUM.

Delirium is a form of mental derangement which only differs from insanity in being acute and temporary, and in being referable to some definite morbid condition. It usually arises from one of the following causes:—(a) inflammation or disease of the brain; (b) fever, especially in connection with specific infection; (c) toxic conditions, such as alcoholism and uræmia; or (d) profound exhaustion. It is characterised by hallucinations, illusions, and delusions. An hallucination is a sense-impression arising independently of any external object corresponding to it. An illusion is an erroneous conception of external sense-impressions, so that an object appears different from what it really is. Delusions are false notions and ideas which have no necessary reference to sense-impressions. Delirium may

be quiet or active. The former is characterised by hallucinations, which dominate the patient's mind, so that he talks incoherently, and as a rule in low muttering tones. In active delirium, the perverted mental processes are more energetic, and the patient tries to act in accordance with them. There is nothing, however, in the character of delirium to suggest its cause, except perhaps in acute alcoholic delirium; and its significance lies mainly in the symptoms with which it is associated, such as tremor, high temperature, the presence or absence of headache, and so on. It should be remembered that delirium is readily induced in alcoholic and senile subjects; and also that it often masks the symptoms of acute local disease.

VERTIGO.

Vertigo or giddiness implies a feeling of instability associated with involuntary movement of the body or a sensation of movement either of the body or of surrounding objects. It implies a disorderly action of the nervous centres concerned in maintaining equilibrium, of which probably the cerebellum is the most important.

Vertigo may be produced by direct disturbance of the equilibrating centres from organic disease, sudden variations in the blood-supply, slowness on the part of the vascular walls to adapt themselves to sudden alterations in posture, or toxic conditions from abuse of alcohol, tea, or tobacco, from certain drugs, or from auto-intoxication, as in uræmia or gastro-intestinal disturbances. On the other hand it may be due to some interference with the centripetal impressions from the eye, ear, muscles, or skin, which regulate the action of the equilibrating centres. Lastly we meet with vertigo as an essential feature of minor epilepsy, or forming the "aura" of severe epileptic attacks.

From most of these causes, vertigo does not amount to more than a vague feeling of unsteadiness. Sometimes,

however, it is distinctly paroxysmal and definite (the movement or sense of movement being constantly in one definite direction); and in these cases it is almost always due to aural disturbance (*labyrinthine vertigo*) or epilepsy.

Aural or labyrinthine vertigo (Menière's disease) may result from any aural disorder which directly or indirectly affects the labyrinth of the internal ear. It is characterised by tinnitus, deafness, vertigo, and is frequently associated with nausea or vomiting. The attacks, though often severe, are not dangerous. The paroxysm gradually passes off, but some tinnitus, giddiness and disturbance of the stomach generally persists for a few days. Recurrence is frequent; but after a while the symptoms usually disappear, with the exception of deafness, which is commonly permanent.

Epileptic may be distinguished from aural vertigo by loss of consciousness, and quick complete recovery. There is no giddiness during the intervals, and there is no persistent tinnitus. If accompanied with convulsive movements, of course the diagnosis is easy.

INSENSIBILITY—STUPOR—COMA.

These terms are used to express different degrees of functional incapacity, beginning at the cerebrum, and characterised at the outset by loss of consciousness, perception, and voluntary motion. In this stage, automatic and reflex movements continue. Afterwards the subordinate ganglionic centres of the brain become involved, and the capacity for automatic movements becomes progressively limited. In still more serious cases the loss of function extends to the medulla (which is the seat of the centres presiding over the important functions of circulation and respiration amongst others), and death ensues.

Pathologically, stupor and coma may be induced by

disturbance of the cerebral circulation (*e.g.* extreme congestion or anæmia); deficient oxygenation of the blood (*e.g.* asphyxia); toxic conditions (*e.g.* uræmia); or by inflammation, structural disease, or injuries of the brain.

LUMBAR PUNCTURE.

A few words are necessary with regard to this matter, as the results of lumbar puncture may have considerable diagnostic value, revealing, as it does, the nature of the cerebro-spinal fluid. In its performance, the patient should lie on his side with the back as fully flexed as possible. The skin should be sterilised, and a hollow steel needle should be introduced forwards, and a little upwards and inwards, at a point on a level with the crests of the ilium (between L 3 and L 4), about half an inch from the middle line. In health the cerebro-spinal fluid is scanty, and only escapes drop by drop after puncture. It is clear, sterile, and (on centrifuging) deposits very few lymphocytes. In tubercular meningitis it is abundant, slightly turbid, and contains many lymphocytes, and sometimes a few tubercle-bacilli. In septic meningitis it is also abundant, often offensive, turbid or purulent, yielding a deposit of polymorphonuclear leucocytes containing pyogenic organisms. It remains clear in cerebral abscess, and thus affords useful evidence in the differential diagnosis of this condition.

SUB-SECTION I.

DIFFUSE DISEASES OF THE NERVOUS SYSTEM.

CEREBRO-SPINAL MENINGITIS.

Meningitis is a generic term applied to any meningeal inflammation. It often happens, as the result of surgical injury, that all the membranes—dura mater, arachnoid,

and pia mater—are equally inflamed ; but as the result of disease this is not so. In one group of cases the inflammation chiefly involves the dura mater (*pachymeningitis*), and usually depends upon disease of the cranial bones ; while in another and much larger group, the pia mater and arachnoid are the seat of inflammation (*leptomeningitis*). Apart from surgical disease, pachymeningitis is quite rare and does not need consideration here. Leptomeningitis may be acute or chronic.

ACUTE LEPTOMENINGITIS.

Acute leptomeningitis may be either primary or secondary. Apart from cerebro-spinal fever (*cf.* p. 84) and basic meningitis of children which is closely allied to it, the primary form is practically always of pneumococcal origin. By far the most frequent and important variety of secondary meningitis, is that which is due to tuberculosis ; but it may also occur from trauma, middle-ear disease, or infective fevers, especially pneumonia and septicæmia.

General Morbid Anatomy.—In some cases the anatomical changes are limited to certain parts of the meningeal surface, such as the convexity or base of the brain, whilst in others they are more or less generally distributed. The membranes are congested, more opaque than normal, and generally covered with a layer of greenish exudation of lymph, especially along the course of the vessels. The subarachnoid space and ventricles often contain an excess of fluid or actual pus.

General Symptoms.—There may be premonitory symptoms in the form of headache, giddiness, and nervous irritability, or of some disturbance of general health. The early stage of the disease is characterised by cerebral irritation and excitement, followed by a stage in which cerebral function is progressively lost as the result of brain compression and toxæmia.

The leading symptoms of the irritative stage are headache, vomiting, delirium, and fever, which develop acutely. The pulse is usually slow at first, and often irregular in force. Breathing is quickened; and both pulse and breathing are subject to irregularity of rhythm. There is general increased sensitiveness to afferent impressions of all kinds, accompanied with muscular twitching, rigidity of the neck and back (occasionally accompanied with retraction), and sometimes convulsion. Kernig's sign is often observed, *i.e.*, when the thigh is fixed to a right angle with the trunk as the patient lies on his back, there is marked resistance to the extension of the knee as a result of flexor spasm. In the course of a few days the acuter symptoms abate, the patient becomes dull and apathetic, tosses about restlessly, and takes food with increasing difficulty. When the base of the brain is affected there may be paralysis of one or more of the cranial nerves. The pupils may be unequal, and optic neuritis frequently occurs in basal cases. From this point the patient rapidly gets worse, and becomes unconscious. Convulsions may recur from time to time, and often form the closing scene of a disease which is generally fatal, after a duration of a few days to a fortnight. On lumbar puncture, the fluid escapes under pressure, is turbid, and contains many cells most of which are polymorphonuclear, together with the organisms to which the disease is due.

TUBERCULOUS MENINGITIS.

This form of meningitis is most common in children between two and five years of age, but adults are by no means exempt. It is usually secondary to tuberculous disease of the bronchial or mesenteric glands. In adults it is generally a sequel of pulmonary phthisis. The anatomical changes, which are closely analogous to those of acute meningitis in general, are mainly observed at the

base of the brain, at the optic chiasma and adjacent parts. Miliary tubercles are seen in the exudation. The ventricles become distended with fluid, by which the convolutions are flattened against the skull. The process generally extends to the spinal meninges.

Symptoms.—As the disease occurs in children, it is commonly preceded by a variable period of disturbed health, characterised by restlessness, irritability, lack of interest in play or lessons, loss of appetite, emaciation, constipation, and disturbed sleep. The actual onset is marked by headache and vomiting, with some rise of temperature. More rarely it sets in with convulsion. The headache is often so severe that the child shrieks out with pain, and holds its head with the hands. Vomiting is frequent, and occurs independently of food. Pyrexia is irregular, seldom runs high, and may be so moderate as to be overlooked. The pulse is generally slow, and irregular both in force and rhythm. The child is very restless, and intolerant of any interference. The pupils are more often contracted, and a squint may often be noted. The bowels are invariably costive. Kernig's sign is nearly always present and the plantar reflex is of the extensor type. The fluid withdrawn on lumbar puncture is abundant, clear or slightly turbid, with cells of mononuclear type. Tubercle bacilli as a rule are very scanty, and can only be found after careful centrifuging.

In the course of about a week, the child becomes drowsy, dull, and restless; the vomiting is less urgent or ceases; the pulse is slower and often irregular; food is only taken with difficulty; the bowels are confined; and the abdomen is retracted. The pupils now dilate, and optic neuritis is often present. Local paralyses are not uncommon, especially of the sixth and facial nerves. As the case proceeds, retraction of the head, grinding of the teeth, tonic spasm of one or more limbs, muscular twitchings, or general con-

vulsions may be observed. Coma now gradually supervenes, wasting becomes extreme, and the child may drag on in this state for many days. The temperature falls, sometimes to subnormal, though it may rise abruptly just before death. The average duration of the disease, which is almost always fatal, is from two to four weeks. Some cases run their course more rapidly.

POSTERIOR BASIC MENINGITIS.

This affection, as it occurs in this country, usually affects infants under the age of one year, and is generally regarded as a modified type of cerebro-spinal fever. As its title implies, the inflammation is limited to the posterior part of the base of the brain.

Apart from the earlier age at which it occurs, the symptoms differ from those observed in tubercular meningitis. It begins abruptly with vomiting or convulsions. The child is in great pain, and screams if moved. He lies on his side, with *retraction of the neck*, the limbs flexed, and the spine often arched backwards. Squint and optic neuritis seldom occur, but blindness is frequent, though the pupils may remain active to light. Intolerance of sound is also often present, but deafness is rare. Vomiting is a much more prominent feature throughout, than in tuberculous cases. The temperature rises rapidly at first, but its subsequent course is very variable. Periodic exacerbations of fever are often observed. As in cerebro-spinal fever, erythematous or herpetic eruptions are common. The results of lumbar puncture are those of acute meningitis in general.

The disease may prove fatal in three or four days; or it may become chronic, and the child may ultimately die with extreme marasmus; but a fair proportion of cases recover in the course of a few weeks.

GENERAL DIAGNOSIS OF ACUTE MENINGITIS.

The recognition of meningitis is often very difficult, and its thorough consideration would occupy far more space than is here available. Thus in secondary cases it may be most puzzling to distinguish meningeal symptoms from those of the associated disease; and the onset of acute specific fevers, especially in young children, is frequently marked by nervous symptoms very suggestive of meningitis. Infants also may develop signs of brain-irritation as the result of marasmus or severe diarrhœa; but the diagnosis will be assisted by the history of the case, depression of the fontanelles, and absence of any focal brain symptoms. Acute meningitis differs from typhoid fever in the irregular course of the temperature; the retracted abdomen; Kernig's sign; the absence of diarrhœa, splenic enlargement, and rose-spots; and the negative response to Widal's test.

In distinguishing the different forms of meningitis from each other we have—apart from careful observation of signs and symptoms—to rely mainly upon the family and personal history of the case, the age of the patient, the mode of onset, the possibilities of septic infection and the evidence of disease elsewhere—in the glands, the lungs, and the ears. But in spite of care, mistakes will occasionally arise.

CHRONIC MENINGITIS.

Chronic meningitis is met with in certain cases of chronic alcoholism, tertiary syphilis, and organic cerebral or spinal disease, but is usually of little clinical interest in itself, apart from the condition with which it is associated. It is characterised by thickening and opacity of the pia-arachnoid, giving rise sometimes to adhesions in which one or other of the cranial or spinal nerves may be involved.

DISSEMINATED, OR INSULAR SCLEROSIS.

Sclerosis, characterised by increase of the neuroglia and connective tissue of the central nervous system, may occur from a variety of causes. It occurs in the neighbourhood of any inflammatory lesion; and is the usual signal of neuronie degeneration, whether due to injury, toxic conditions, or interference with blood-supply. But apart from these obviously secondary forms of sclerosis, cases are of common occurrence in which, though perhaps not primary, sclerosis is the dominant feature, and of these, disseminated or insular sclerosis is the most important.

Disseminated or insular sclerosis is characterised anatomically by islands of sclerosis scattered about in the central nervous system. The affected areas are irregular in shape and size, sharply defined, of a greyish colour, and more often of firm consistence. They consist of local over-growths of neuroglia; and the nerve-fibres which pass through them, though they lose their medullary sheaths, retain their axis-cylinders unbroken for a long time. The patches are most common in the pons, basal ganglia, and spinal cord, but may occur in any part of the central nervous system. The peripheral nerves are never affected, the optic nerves (which are not true peripheral nerves) being the only exception. It is uncertain whether the process begins in the neuroglia or the nerve-structures, but the evidence is in favour of the latter view.

It is essentially a disease of early adult life, occurring most often between 16 and 30 years of age. Previous attacks of infective disease, especially influenza, appear to favour its occurrence, also nervous over-strain, and exposure to cold. It appears to have no causal relation to alcoholism or syphilis.

SYMPTOMS.—From the above remarks it is obvious that

the symptoms will vary according to the distribution of the sclerotic nodules. In any case the onset is very slow and insidious, and it is generally a long time before the nature of the disease is definitely declared. Among the early symptoms, weakness of the lower limbs, especially after exercise, slight ataxy (not materially increased on closing the eyes), definite increase of knee-jerks, numbness or tingling of the extremities, giddiness, and slight tremor are most frequent.

When once the disease is established, the typical symptoms are more or less paraplegia of a spastic type, intention or action tremor, staccato speech, and nystagmus. Tremor on attempted voluntary movement (intention tremor) is a very marked feature, but when the patient is at rest or asleep it is absent. If he takes up a glass of water and tries to drink, the rhythmic agitation becomes more and more violent as the glass approaches the mouth. Writing, too, is almost impossible. Speech is generally slow, staccato, and hesitating, but sometimes it is explosive in character. Nystagmus consists of rapid jerking rhythmical movements of the eyeball from side to side, which are developed on forced deflection of the eyes to the right or left. Spastic paraplegia is rarely absent, the knee-jerks are exaggerated, ankle-clonus is present, the plantar reflexes are extensor. Anæsthesia is uncommon, but numbness, tingling, and sensations of heat and cold are complained of. There is early affection of the sphincters, precipitate micturition followed by incontinence or retention of urine being common. Constipation is the rule, and there is sometimes difficulty in the act of defæcation. Primary optic atrophy with dimness of vision is common and though unilateral at first, both eyes are ultimately affected. Defective vision may occur without obvious change in the disc. The pupils are often unequal and sluggish to light. Some degree of mental impairment is

generally present sooner or later. During the course of the disease, sudden apoplectiform attacks have been recorded.

Diagnosis in the early stage is sometimes very difficult ; and the conditions most likely to cause confusion are hysteria, sub-acute combined degeneration of the cord, and general paralysis. There is reason to believe that recovery may sometimes take place, but as a rule the prognosis is bad. The general nutrition suffers ; bed-sores and cystitis are apt to occur ; the patient becomes highly emotional, and the arm-tremor may be so severe that he is unable to feed himself ; and, after a course of five to twenty years, death occurs from asthenia or some complication.

GENERAL PARALYSIS OF THE INSANE.

Although the plan of this work does not include mental diseases, it seems desirable to refer shortly to that general and progressive form of nervous break-down known as *general paralysis*, or *paralytic dementia*. It is a disease which begins with mental perversion, and a steady degradation of nervous function, and ends with complete dementia and general paralysis. It is most frequent in men between 30 and 50 years of age. Nervous worry, over-strain, and syphilis are the principal causes. The pathology is obscure.

The anatomical changes are general, affecting the brain and cord with their membranes, and the peripheral nerves. There are evidences of more or less diffuse chronic meningitis. The brain is hyperæmic, its convolutions are shrunk, and its consistence is usually softer than normal. The cord is chiefly softened, but sometimes indurated, and degeneration of the posterior columns is not uncommon. The histological changes essentially consist of atrophy of the nerve-elements, and hyperplasia of the neuroglial

and connective tissue elements. The cerebro-spinal fluid is in excess, with increased albumen, and leucocytosis.

The *symptoms* vary considerably in different cases, but in the ordinary form the disease begins with mental and moral disturbance, such as irritability, restlessness, tendency to extravagance, failure of memory, and carelessness of appearance and manner. After a variable interval, signs of muscular weakness appear, with tremor of the lips, tongue, or hands, on voluntary movement. The pupils react on accommodation, but are sluggish or fail to respond to light, and are often unequal. Speech becomes blurred and indistinct, special difficulty occurring with labial explosive and lingual consonants, as in the word "parallelogram." From this point onwards, the history of the case is one of progressive mental and physical deterioration. On the one hand, the patient becomes more and more incapable of mental exertion; and delusions, usually of an extravagant and optimistic kind, develop. Emotional, or even maniacal outbursts are of frequent occurrence, and the patient finally drifts into complete fatuity. On the other hand, muscular weakness extends over the whole system, the faculty of articulation is lost, general paralysis supervenes; bed-sores develop, and in the last stage the patient lies perfectly helpless. Epileptiform and apopleciform attacks are common. Remarkable remissions in this downward course are occasionally met with. The affection is always fatal, and its average duration is about three years.

The most important factors in *diagnosis* are the history of the case, especially with regard to syphilis, the age of onset, tremor of the tongue and lips, defective articulation, inequality and abnormal behaviour of the pupils, associated with progressive mental and motor failure. The knee-jerks are generally increased except in tabetic forms, and the plantar reflex is commonly extensor.

SUB-SECTION II.

DISEASES OF THE BRAIN.

CEREBRAL ANÆMIA AND HYPERÆMIA.

Very moderate variation in the intra-cranial blood-content is sufficient to give rise to nervous disturbance. Under normal conditions, however, such variation is obviated by the fact that anæmia of the vaso-motor centre, in the medulla—whether produced directly by arterial under-filling or loss of blood, or indirectly by increase of venous pressure—causes constriction of the arterioles in the splanchnic area, with rise in the general blood-pressure, and increased flow of blood to the brain. But when the vaso-motor centre is exhausted by debility, or semi-paralysed by an anæsthetic, such adjustment is prevented; and, under these conditions, mere change in posture may suffice to cause discomfort and even danger, by permitting serious variation of the intra-cranial blood-content. Analogous results may follow on the sudden diminution of pressure in the splanchnic area by too rapid removal of an ascitic effusion. Yet again, if the heart be too weak to keep up an adequate supply of blood to the brain, or in the event of temporary arrest of its action, as the result of shock or heart-block, serious symptoms may occur from cerebral anæmia. Owing to the number, size, and freedom of anastomosis of the cerebral sinuses and veins, marked venous congestion is only likely to occur when *all* are simultaneously affected as with strong expiratory efforts against a closed glottis. Thus straining at stool, severe paroxysms of coughing, or lifting heavy weights may not only give rise to nervous disturbance, but to actual hæmorrhage into the brain, by raising intra-cranial blood-pressure.

Symptoms.—Since arterial under-filling and mechanical venous congestion are both characterised by deficient blood-supply, the symptoms in each case are much the same.

In *sudden* anæmia of the brain, the main symptoms are drowsiness, giddiness, failure of special senses, singing noises in the ears, dilated pupils, numbness, mental confusion, and nausea. If more severe, there may be complete insensibility, convulsions, and general collapse, passing into fatal coma. When the anæmia is *slowly produced and persistent*, we meet with general mental irritability, restlessness, troublesome insomnia, inaptitude for mental work, loss of memory, vertical headache relieved by the recumbent posture, together with a sense of heavy pressure on the top of the head.

A condition, sometimes termed *hydrocephaloid*, which occurs in infants, usually as a sequel to exhausting diarrhoea, has been ascribed to cerebral anæmia. It is characterised by drowsiness and general collapse gradually merging into coma, with pallor, cold extremities, *depressed* fontanelles, and retracted head.

ACUTE ENCEPHALITIS AND CEREBRAL ABSCESS.

Inflammation of the brain may be either suppurative or non-suppurative. The former, when local, results in a cerebral abscess, and may arise from injury, suppurative affections of the middle ear, disease of the cranial bones, or (more rarely) from secondary infection from purulent disease elsewhere, especially in the lungs. When due to a local cause the abscess usually occupies an adjacent part of the brain; thus in middle-ear disease the temporo-sphenoidal lobe or lateral lobe of the cerebellum is most frequently involved. Such abscesses are usually single,

and are sometimes of large size. They may cause death by the increased pressure to which they give rise, or they may burst into one of the ventricles or on to the surface of the brain. A moderate degree of local meningitis is not uncommon. The abscesses of pyæmia are multiple and of small size, scattered promiscuously throughout the substance of the brain.

Cerebral abscess may be assumed to have occurred when, after some cranial disease or injury or in the course of a chronic otorrhœa, inflammatory and febrile symptoms supervene, accompanied with localised headache, vomiting, persistent drowsiness deepening into coma. Somewhat later the temperature often falls to normal, and the pulse becomes slow. Optic neuritis is frequent, but is often late in development. Convulsions are not common, but paralysis of some kind occurs in about half of all cases, probably as the result of brain-pressure. Cerebellar abscess may be attended with a staggering gait, deviation or retraction of the head with rigidity of some of the muscles of the neck, and irregular squint. In certain cases, cerebral abscess may be entirely latent for long periods, until the sudden occurrence of severe headache, vomiting, and fever reveals the nature of the disease. Apart from a history of injury or ear-disease, the diagnosis may be very difficult. Unless relieved by operation, the termination of intra-cranial suppuration is fatal. In pyæmic abscess, the symptoms are merged in those of the primary disease.

Acute non-suppurative encephalitis is a much rarer condition, affecting the grey matter of the brain (*polio-encephalitis*). It is characterised anatomically by vascular engorgement, with minute hæmorrhages, thrombosis, and exudation of leucocytes. No doubt more or less inflammation of brain tissue accompanies all forms of meningitis; but the condition now under consideration is a substantive affection of obscure origin, strictly analogous to the more

familiar condition affecting the cord (*polio-myelitis*). It may attack the cortex of the brain (*P. superior*), in which case hemiplegia is the most common result, explaining—as is believed—some of the cerebral palsies of young children. Or again, it may attack one or more of the nuclei of cranial nerves (*P. inferior*), such as the oculo-motor nerve, causing ophthalmoplegia; or facial nerve, causing facial paralysis. More rarely, the cerebellum is affected (*P. cerebelli*), causing acute ataxia of a cerebellar type. The tendency to recovery is much greater than in other acute cerebral lesions.

CEREBRAL HÆMORRHAGE. APOPLEXY.

Cerebral hæmorrhage, or apoplexy, denotes rupture of a vessel in the brain. It is for the most part a disease of middle life, occurring most frequently from 40 to 50 years of age, and more commonly in men than women. It is primarily due to weakening of the arterial coats by disease, of various kinds; but is favoured by a high blood-pressure, whether the result of disease, effort, or excitement. The etiology therefore is, in the main, that of arterial disease (p. 322).

ANATOMICAL CHARACTERS.—When the hæmorrhage is small the nerve fibres are merely pushed asunder with little pressure on surrounding parts. On the other hand, in large hæmorrhage the brain tissue is ploughed up, a potential cavity is formed of roundish shape with ragged walls, and filled with blood which soon clots, and considerable pressure may be exerted upon the brain. The hæmorrhage may extend into the ventricles, or, more rarely, through the cortex of the brain into the arachnoid cavity. Rupture is most frequent in one of the deep branches of the middle cerebral artery supplied to the corpus striatum and internal capsule; and less frequently in the optic

thalamus. Hæmorrhage into the cerebellum, crus cerebri, pons varolii, or medulla is relatively uncommon.

Should the patient survive, the *clot* shrinks and breaks up into a reddish-brown fluid. The cavity gradually becomes smaller, and its boundaries more defined. In small hæmorrhages, complete absorption may take place, leaving only a pigmented scar. In other cases a permanent cyst may be found, with pale fluid contents. Crystals of hæmatoidin are often met with. The more important secondary effects upon the surrounding nervous structures are :— (1) flattening of the convolutions and cerebral anæmia, from pressure ; (2) local inflammation due to damage of the brain substance ; and (3) secondary degeneration of the damaged motor tract.

SYMPTOMS.—The symptoms of cerebral hæmorrhage may be conveniently considered under two heads : (1) the “*apoplectic stroke*,” which is due to the sudden functional disturbance of the brain as a whole, from shock and pressure ; and (2) *paralysis*, which is directly due to the local injury.

The essential feature of an apoplectic attack is coma, the profoundness of which is related to the amount, suddenness, and seat of the hæmorrhage. Premonitory symptoms are rare ; and, when present, are not characteristic. The onset may be sudden or gradual. An attack rarely occurs during sleep, but rather at a time when the circulation is vigorous, as in the early part of the day, or during exertion of some kind. In a typical case of the ordinary type there is complete loss of consciousness accompanied with a full bounding pulse, and slow, deep, or stertorous breathing, with puffing of the cheeks. Convulsions are uncommon. The face is dusky, the skin is often bedewed with sweat, and the head and eyes may deviate to one side—usually *towards* the side of the brain affected. In severe cases all reflexes and tendon-jerks are abolished,

the limbs lie flaccid and helpless, and involuntary evacuation of the bowels may take place, but as a rule the urine is retained. The temperature falls at first in proportion to the completeness of the coma, but within a day or two, it rises as the result of reactive local inflammation, and there may be more or less febrile disturbance for a week or longer. After a variable time consciousness may gradually return; but often, as the result of increasing intra-cranial pressure, the breathing becomes more and more laboured, and death occurs from asphyxia.

In more favourable cases, consciousness returns, and then the paralytic features become more obvious. The distribution of paralysis of course varies according to the seat of hæmorrhage; but as the latter usually involves the middle third of the internal capsule, as already pointed out, it is therefore attended by paralysis *on the opposite side of the body*, of the arm and leg, of the lower facial muscles, and usually of the muscles which protrude the tongue (*hemiplegia*). The detailed features of hemiplegia have been already described (p. 495). Hæmorrhage into the left half of the brain is generally accompanied with *aphasia*; but more or less difficulty of articulation is of common occurrence, quite apart from aphasia. *Hemi-anæsthesia* is associated with hemiplegia when the hinder part of the internal capsule is implicated. As consciousness is recovered the reflexes begin to return. On the paralysed side the knee-jerk is exaggerated, and the plantar reflex (as soon as it can be obtained) is of the extensor type; but many of the superficial reflexes are languid for a long time. The sphincters are not affected. Motor power in the limbs may also return in favourable cases, the leg recovering before the arm as a rule; but more often, recovery is imperfect, and freedom of movement is further hindered by slowly increasing spasticity on the paralysed side.

Escape of blood into *the ventricles* only occurs with large hæmorrhages. It may be attended with initial rigidity, but it is soon lost in the rapidly deepening coma, and gives place to complete general flaccidity, with early death.

Hæmorrhage in *the pons* when quite small may give rise to a crossed hemiplegia of some kind, such as facial paralysis on one side, with paralysis of the limbs on the opposite side. But far more often the coma is too profound, and the duration of life too short, to make any observations on this point. The pupils are contracted to pin-points, and the temperature rapidly rises to a high grade.

Cerebellar hæmorrhage is extremely rare and demands no special description here.

The *clinical history* of cerebral hæmorrhage varies greatly according to its seat, extent, and rapidity of occurrence.

1. Usually profound coma supervenes, with or without premonitory symptoms, in which the patient dies in the course of one to several hours—*never instantaneously*.

2. Less frequently the attack begins with coma, and after some hours consciousness gradually returns; but hemiplegia with or without aphasia remains, and persists for the remainder of life.

3. Partial recovery may occur as before, but owing to fresh hæmorrhage the coma returns and proves fatal. This frequently means a sudden extension of hæmorrhage into the lateral ventricle.

4. In cases where the hæmorrhage is slow, and steadily progressive, symptoms of paralysis may precede coma.

In conclusion, certain secondary phenomena deserve mention. 1. As a result of *local inflammation* set up by the hæmorrhage, febrile symptoms attended with headache, restlessness, mental confusion, or even delirium may be observed. These symptoms (if present) usually continue for four or five days, and then pass off. 2. *A peculiar permanent change of manner* with loss of memory and emotional

perversion is apt to occur after cerebral hæmorrhage. 3. *Bed-sores* sometimes appear on the buttock of the paralysed side, but they are not common. 4. In cases which survive the early stages, spastic rigidity of the paralysed muscles gradually develops, and may be accompanied with local pains (*late rigidity*). It depends upon secondary descending degeneration of the motor tract. 5. *Other nervous symptoms* may occur, such as choreiform movements, delirium, mania, or dementia, but are relatively rare.

The *diagnosis* of apoplectic coma from alcoholism, uræmia, and opium-poisoning is at times very difficult. *Apoplexy* is for the most part a disease of late middle life; the skin is moist and warm; if the coma is profound it is usually rapidly attained; evidence of hemiplegia can generally be obtained; deviation of the head and eyes is especially characteristic when present; and the temperature is depressed at first, and then rises. In *alcoholism*, a history of intemperance may be obtainable; there is no paralysis or convulsion, and the coma is rarely very profound. In *uræmia*, the urine is always albuminous; the skin is cool and dry; paralysis is rare; convulsions are common; and the temperature is persistently depressed. In *opium-poisoning*, we are guided by the history of the case, a low temperature with moist skin, and marked equal contraction of the pupils—the latter not accompanied with pyrexia, as in pontine hæmorrhage. After all said and done, mistakes may easily be made, and a golden rule is, “never dismiss a case from observation while the diagnosis is uncertain.” The diagnosis from embolism and thrombosis will be considered when describing these diseases.

PROGNOSIS.—The chief unfavourable signs are these: Very profound coma, duration of coma beyond twelve hours, irregularities of breathing, and a high or progressively rising temperature. Much, too, depends upon the

seat of mischief. Thus, hæmorrhage into the ventricles is always fatal, and into the pons is nearly always so. Other things being equal, albuminuria is of unfavourable import.

MENINGEAL HÆMORRHAGE.

Meningeal hæmorrhage is usually of traumatic origin, and may be on either side of the dura mater. Sub-dural bleeding may also occur in hæmorrhagic diseases such as purpura or pernicious anæmia. But a more important variety is that which not uncommonly takes place on the brain-surface of an infant, more often spreading from the longitudinal sinus, as the result of long and difficult birth. Though instruments may have been used in delivery, the hæmorrhage is due to the conditions leading to their employment, rather than to the instruments themselves. The adjacent cortex of the brain is thus damaged or disturbed. In severe cases, the infant may be still-born, or die very shortly after birth ; while slight cases may recover completely and permanently. On the other hand important and persistent symptoms may arise as the result of secondary changes occurring in adjacent brain-substance, characterised for the most part by paralysis (*birth-palsy*).

There may be an attack of convulsions or paralysis at the outset, but nothing may be noted until the time when the child should begin to walk, when the legs are found to be stiff, so as to interfere with walking. But slight stiffness, and increased knee-jerks may be observed much earlier, if looked for. By two or three years of age the symptoms are fully developed. They are generally bilateral, and the legs are more often affected than the arms, causing a spastic paraplegia (*diplegia*). In a severe case the child cannot stand or walk ; and, if held up, the legs are flexed, stiff, and often crossed ; and the feet are extended. The child may be able to crawl fairly well ;

and in a majority of cases, he ultimately learns to walk, but more or less awkwardly. Irregular movements may develop, especially in the hands (when the arms are affected), a condition known as *athetosis*. Persistent contractures may also occur. The child is usually backward in talking, and the mental condition is often defective. Little can be done by way of treatment in chronic cases, but removal of clots by operation at the outset has been successful in certain cases.

CEREBRAL EMBOLISM AND THROMBOSIS— CEREBRAL SOFTENING.

CEREBRAL EMBOLISM is caused by plugging of one of the cerebral arteries—almost always the *left middle cerebral* artery. The embolus usually takes the form of a small vegetation or particle of fibrin which becomes detached from the heart or aorta, and is swept into the current of the circulation. It occurs most frequently in valvular disease of the left heart, especially mitral stenosis, and is therefore more common in early adult life. Pyæmic abscesses result from infective forms of cerebral embolism, but they are not included in the present description.

CEREBRAL THROMBOSIS generally results from arteriosclerosis or syphilitic arteritis of cerebral vessels, which leads to a local coagulation of blood within them. A languid circulation favours its occurrence, and therefore is more common during sleep, or when a patient is tired after a day's work. Thrombosis of syphilitic origin generally occurs between twenty-five and forty years of age, but otherwise its frequency increases with age.

Both embolism and thrombosis have this feature in common—that *the supply of blood is cut off* from that part of the brain to which the obstructed vessel is distributed; and that consequently, if collateral circulation is not

quickly established, and if death does not intervene, it undergoes *softening and necrosis*. The colour of the affected area varies according to the amount of blood-pigment present in it, and the change it has undergone. It is more often yellowish (*yellow softening*). Small areas of softening may be completely absorbed, and only a small scar may be left behind; in other cases absorption is not complete, and a cyst is formed, containing a clear fluid. Neither embolism nor thrombosis raises the intra-cranial pressure, as happens in hæmorrhage.

SYMPTOMS.—In embolism *premonitory symptoms* are wanting; but in thrombosis, owing to the general arterial disease with which it is associated, premonitory symptoms are often observed, such as headache, giddiness, loss of memory, numbness, tingling or transient weakness of the limbs on one side, or slight aphasia.

In *embolism* the onset is sudden, and generally attended with loss of consciousness. The patient may possibly pass directly into fatal coma; but as a rule, the loss of consciousness is of short duration, or it may not occur at all. As this passes off, the patient is found to be paralysed. Of course, the paralysis will vary according to the seat and extent of the obstruction; but as already pointed out, by far the most frequent seat of embolism is the left middle cerebral artery, which supplies the greater part of the corpus striatum, the internal capsule, the anterior half of the optic thalamus, and nearly the whole of the antero-lateral region of the brain. Hence there is usually right hemiplegia with aphasia.

In *thrombosis* a sudden onset is exceptional. Premonitory symptoms are apt to occur towards the end of the day, and the patient perhaps wakes up next morning more or less hemiplegic. The hemiplegia, in any case, usually reaches its height well within twenty-four hours. The patient is dull and sleepy, and may be temporarily un-

conscious ; but if so, it is seldom complete. The paralysis is generally obvious, with increased tendon-jerks and extensor plantar reflex on the affected side. In severe cases there may be retention of urine, or the sphincters may be relaxed unconsciously.

Though the actual attack of embolism or thrombosis is survived, the paralysis more often persists in some degree, and is accompanied with other indications of cerebral softening, such as persistent frontal headache, loss of memory, and general mental enfeeblement passing on to complete dementia. Bed-sores on the buttock may also appear, as after cerebral hæmorrhage. Similarly, late rigidity is of common occurrence, and death ultimately occurs from asthenia or some complication. In other cases, especially in thrombosis, the patient becomes slowly more comatose, and dies after an interval of some days, or a week or two. Lastly, there are cases, especially those of syphilitic origin, which if properly treated, may completely recover. It should be recognised that a patient may suffer from repeated attacks of thrombosis, due to successive implication of other cerebral arteries.

With regard to the diagnosis between cerebral hæmorrhage, embolism, and thrombosis, attention should be given to the following points. Hæmorrhage is most common from 40 to 60 years of age ; the onset is generally rapid, and the coma is profound ; the rectal temperature is lowered within an hour of onset to a subnormal point, rising in the course of two to three hours to about 101° , or higher in fatal cases, and often associated with albuminuria, vascular rigidity, and hypertrophy of the left ventricle. Embolism is of sudden onset ; usually occurs in those under 40 years of age ; is associated with valvular disease, aneurism, or puerperal thrombosis ; loss of consciousness is more transient, owing to the absence of brain-pressure ; and the rectal temperature rises temporarily in two or

three hours without previous depression. Thrombosis may occur at any age; the onset is gradual; premonitory symptoms are common; there is usually no loss of consciousness; it is associated with a low blood-pressure and with evidence of syphilitic or other forms of arterial degeneration, and the temperature is little disturbed.

THROMBOSIS OF THE CEREBRAL SINUSES.

Thrombosis of the cerebral sinuses may be either *simple* or *infective*. In the former we have only to do with the effects of local vascular obstruction; while in the latter, the local trouble is associated with a general pyæmia.

Simple thrombosis more often affects the longitudinal sinus, and is most common in infants as the result of marasmus. The symptoms are by no means characteristic; but if, under the circumstances indicated, convulsions take place, accompanied with restlessness, vomiting, and perhaps some œdema of the scalp, this condition may be strongly suspected.

Infective thrombosis more often affects the lateral sinus; and, in the great majority, is secondary to disease of the middle ear. It may occur at any age. The most prominent symptoms are severe headache, vomiting, repeated severe rigors, profuse sweating, and irregular temperature with marked variations. Locally there may be pain and tenderness, with some œdema over the mastoid process. Or the dominant symptoms may be those of an acute meningitis. With knowledge of the existence of a discharge from the ear, and marked local symptoms, the diagnosis may be relatively easy, but otherwise it may be difficult. The prognosis is very grave. If recognised early, some cases may be saved by timely operative interference, the details of which may be found in surgical works.

TUMOURS OF THE BRAIN AND MEMBRANES.

The more important of the new formations to which the brain and membranes are liable are as follows :—

1. *Tubercle*.—This is probably the commonest form of cerebral tumour, especially in early life. It is usually single, and consists of a rounded cheesy mass, resulting from the degeneration of aggregated groups of tubercle. It varies in size from the size of a pea to that of a hen's egg. Its usual seat is on the surface of the brain, or in the cerebellum, and is most frequent in childhood.

2. *Glioma*.—This form of cerebral tumour is most frequent in adults. It arises in connection with the neuroglia, or supporting connective tissue of the brain substance. Its structure is closely allied to that of sarcoma; and it is usual to distinguish a hard and soft variety, according to the predominance of fibres and cells respectively. It may occur in any part of the brain.

3. *Syphilis*.—Syphilitic gummata generally occupy a superficial position in the cerebral cortex or pons, and as a rule are connected with or adherent to the meninges and arteries. They are frequently multiple, vary much in size, and when situated at the base of the brain, the cranial nerves are very liable to become involved.

Other kinds of cerebral tumour are sometimes met with, *e.g.* sarcoma, cancer, myxoma, lipoma, cysts of various kinds, and aneurism; but on account of their rarity, no special description of them is necessary here.

SYMPTOMS.—It may happen that even a large tumour may exist without giving rise to any symptoms whatever during life, especially when its growth has been very slow. In other cases a tumour may exist and cause no symptoms until a few days before death, when convulsions occur followed by a fatal coma.

In describing the symptoms of intra-cranial tumours it is convenient to distinguish between those which are general or diffuse, and those which are focal or localising. The most important general symptoms are *headache*, *giddiness*, *vomiting*, and *optic neuritis*. They depend mainly upon pressure exerted by the growth, and their clinical value, as indicative of organic intra-cranial disease, is related to their presence in combination and their persistence, rather than to their degree. Headache is the most frequent, and may be intense; but whether intense or slight, it is usually more or less persistent, though it may exhibit paroxysmal exacerbations. Its character is variable, and its seat cannot be trusted as an indication of the position of the growth. Vomiting is much less frequent than headache. It is often unaccompanied with nausea. It occurs independently of food, and is apt to be most marked on first moving about on rising in the morning. Constipation is the rule, and the tongue may be coated, and the breath foul. Optic neuritis is present in a large majority of cases and is of greater diagnostic value than any other single symptom of intra-cranial growth. It is often unilateral to begin with; but later on, both discs are usually affected and it is noteworthy that vision may be intact until a late stage. Lastly, general convulsions may take place apart from any direct relation to the seat of the growth.

Focal symptoms should be studied in connection with the functions which belong to the different parts of the brain (p. 534). In tumours of the cortex of the brain, the symptoms differ according to the part affected. They may be irritative or destructive; in other words, they may morbidly excite or abolish the functions of the part affected. Thus in tumours of the motor area, there may be convulsion limited to one limb, or to one side of the body (*Jacksonian epilepsy*), and such convulsion may be followed

by temporary paralysis. Or, again, there may be aphasia, or disturbance of vision or hearing. In tumour involving the *motor tract*, there may be hemiplegia. Tumour at the *base of the brain* is almost certain to involve one or more of the cranial nerves. A growth in the *crus cerebri* is likely to be attended with oculo-motor paralysis with hemiplegia or sensory disturbance on the opposite side (*crossed paralysis*). Tumours of the *cerebellum* do not commonly give rise to paralysis. On the other hand, inco-ordination and giddiness are marked features, and curious paroxysmal attacks of tonic spasm are not infrequent.

Mental disturbance is generally a late symptom and is characterised by gradually advancing imbecility. *Death* usually occurs from convulsions or coma, or a combination of both.

HYDROCEPHALUS.

Hydrocephalus is not a disease in itself, but is a clinical term applied to those cases in which abnormal accumulation of fluid in the ventricles of the brain is the essential feature. It will conduce to clearness, if we reserve it also for congenital cases, in which there is as a rule no readily apparent cause.

The ventricles are distended with a clear limpid fluid, alkaline in reaction, containing only traces of albumen, and varying in amount from one to several pints. As the cranial sutures are not yet united, the effect on the surrounding parts is very striking. The cortex of the brain is greatly stretched and thinned, the outlines of the convolution are lost, and the basic ganglia are flattened. The skull steadily enlarges, the fontanelles and sutures widen, and the bones are thinned.

Symptoms.—More often the child appears healthy at birth, and it is not until some few months later that the

head is noticed to be unusually large. Sometimes, however, convulsions may be the earliest symptom. The enlargement of the head slowly but steadily increases, and assumes a rounded shape. The forehead is round and projecting; the orbital plates become depressed, so that the eyes are prominent, and look downwards; the fontanelles and sutures widen, and may fluctuate on palpation; and the superficial veins are very distinct. The face appears very small by contrast, and wears a curious careworn expression.

The mental condition varies. Intelligence is often surprisingly preserved; but in early and rapidly progressive cases, there is marked deficiency. The limbs are weak, and become gradually spastic, with increase of reflexes. Nystagmus is not infrequent; and, as the result of pressure and tension of the optic tracts and nerves, atrophy of the optic disc may occur, with blindness. The general health is not seriously affected for a long time; but more often wasting sets in, and death takes place from exhaustion—usually within three or four years. Sometimes, however, the condition is arrested, and recovery takes place.

When established, the condition is easily recognised; but in the early stages diagnosis is often uncertain. In all cases, the enlargement of the head requires to be carefully distinguished from that due to rickets. (*Cf.* p. 156.)

Closely allied with congenital hydrocephalus are those cases in which a hydrocephalic condition develops in infancy as the result of posterior basic meningitis, or an intracranial tumour; and where, by reason of meningeal thickenings, about the roof of the fourth ventricle, closing the foramen of Majendie, or by pressure on the veins of Galen, there is interference with the normal drainage of the ventricular cavities.

TOPICAL DIAGNOSIS OF BRAIN LESIONS.

MOTOR CORTICAL CENTRES.—These occupy the ascending frontal (anterior central) convolution—the lower third for movements of the face and adjacent parts; the middle third for those of the arm and hand; and the upper third for those of the leg and foot—all of the opposite side.

SENSORY CORTICAL CENTRES.—The centres for *common sensation* occupy the posterior central convolution, and the falciform lobe on the mesial surface. On the external surface of the brain, the sensory area extends backward into the superior parietal convolution (*form-perception*), and into the inferior parietal (*muscular sense*). The *primary centre for vision* occupies the cuneus of the occipital lobe, and adjacent part; while the *secondary* or *perceptive visual centre* is mainly in the angular gyrus. The *primary centre for hearing* is seated in the middle of the first temporal convolution, and the *perceptive centre* occupies the posterior part of the same. The *centre for smell* is in the hippocampal region.

Lesion of the anterior central convolution will cause spasm or paralysis of the parts which it represents. If it extends backwards sensory disturbance of some kind is likely to be added, varying according to the precise distribution of the lesion.

Lesion of the centrum ovale only gives rise to symptoms when it involves those parts which are in direct connection with the motor or sensory cortex. The remainder is “silent.”

Lesion of the corpus callosum is generally attended with paralysis—at first hemiplegic, and afterwards more or less diplegic, associated with early mental perversion.

Lesion of the internal capsule.—The anterior end is silent; the knee and anterior part of the posterior limbs represent the motor tract, and therefore a destructive lesion will

cause hemiplegia. The posterior third is formed by the sensory tract, and a similar lesion will cause hemianæsthesia.

Lesion of the corpus striatum appears to be attended with weakness of one side, sometimes associated with irritative symptoms. The functions of the part are not well understood, but are probably supplementary to the motor tract.

Lesion of the optic thalamus may also give rise to hemiplegic weakness; but persistent hemianæsthesia, ataxic disturbance, and athetosis are probably more characteristic symptoms.

Lesion of the crus cerebri.—If seated in the crusta, it gives rise to hemiplegia on the opposite side, with implication of the third (and sometimes the fourth) nerve.

Lesion of the corpora quadrigemina.—Probably the most frequent and characteristic symptom is more or less extensive oculo-motor paralysis.

Lesion of the pons.—There may be absence of all symptoms for a long time, but crossed hemiplegia (paralysis of one side of the body, with facial or oculo-motor paralysis on the other) is a usual result.

Lesions of the cerebellum.—The cerebellum may be regarded as a large ganglionic centre interposed (physiologically) between the cortex and the deeper nuclei, for the purpose of rearranging and co-ordinating nervous impulses as they pass to and fro between them. With regard to its influence on sensory impulses, nothing definite is known; but, in cerebellar disease, there may be many different indications of motor disturbance. The most important are tremor, loss of muscular tone, muscular paresis, nystagmus, and staggering, reeling gait (often with a tendency to fall on the side of the lesion). Cerebellar ataxy, unlike that of tabes, does not increase when the eyes are shut, owing to its central origin. Any of these conditions (when present) are most marked on the side of the lesion. Inability to execute movements in rapid succession, such as alternate

pronation and supination of the wrist, may also be observed. In disease of the middle lobe, the head is often retracted. In disease of a lateral lobe, the head may be bent to the side of the lesion, slightly rotated, and looking upward and away from the lesion. This attitude may be well marked in the tonic seizures which sometimes take place in cerebellar disease. From the position of the cerebellum beneath the tentorium, pressure symptoms are common, such as headache, vomiting, and choked disc. Cranial nerves are also sometimes involved by pressure, especially the seventh and eighth.

SUB-SECTION III.

DISEASES OF THE SPINAL CORD.

SPINAL MENINGITIS.

ACUTE SPINAL MENINGITIS, as a separate disease, practically only occurs as the result of surgical injury, or caries of the spine. Otherwise it is associated with acute inflammation of the cerebral membranes, the various forms of which have already been described.

CHRONIC SPINAL MENINGITIS often occurs as a secondary caries affection, in connection with injury, tumours, and tuberculous or syphilitic lesions. A *chronic local serous meningitis* has also lately received some attention. It is characterised by a local circumscribed collection of arachnoid fluid, which by pressure upon the cord, gives rise to symptoms which are indistinguishable from those due to tumour of the cord. Several cases of successful treatment of this condition by operation have been recently reported by Horsley.

Another variety of chronic meningeal inflammation, known as *chronic hypertrophic cervical pachymeningitis*, also claims brief notice. In this affection the dura mater

in the cervical region of the cord becomes greatly thickened and adherent to adjacent parts. In consequence, the spinal cord and its roots at this part become irritated, compressed, and degenerated. Its etiology is obscure, but a history of syphilis is often obtainable. It develops slowly and runs a very chronic course, which may extend over many years. Its progress, broadly speaking, is marked by three stages corresponding respectively to irritation of the local nerve-roots, degeneration of the same, and local compression with degeneration of the cord. Thus we meet first of all with a period of pain and spasm in the parts supplied by the affected roots, secondly a period of peripheral paralyses in the upper limbs, and thirdly a period of spastic paraplegia. It should be distinguished from caries of the cervical spine, progressive muscular atrophy, and peripheral neuritis.

SPINAL HÆMORRHAGE.

Hæmorrhage may take place either into the substance of the spinal cord (*hæmatomyelia*), or into the spinal membranes. Primary hæmorrhage into the cord from disease of the vessels is very rare. Small hæmorrhages are frequent in connection with tumour, myelitis, or syringomyelia, but are of little clinical importance. It is generally of traumatic origin, and limited to the grey matter of the cord. Meningeal hæmorrhage may be either extra-dural or intra-dural. The former is nearly always traumatic, and the latter is apt to occur in children at birth, or as the result of severe convulsion, muscular strain, or bursting of an aneurism at the base of the brain.

Hæmatomyelia is characterised by the sudden onset of motor and sensory paralysis up to the level of the lesion. The legs are often flaccid just at first, with loss of reflexes and knee-jerks. In severe lumbar hæmorrhage this condition may persist, but in cervical or dorsal cases (which are

much more common) the reflexes soon return, and become exaggerated, with paralysis of a spastic type, and more or less dissociated anæsthesia. Not infrequently, the lesion is unilateral, in which case the paralysis is on the same side, and the anæsthesia on the opposite side. Pain is not a prominent feature, and (when present) is limited in extent, and temporary in duration. Death is unusual. More often considerable improvement takes place, but recovery is seldom complete.

Meningeal hæmorrhage, when severe enough to cause symptoms, is characterised by sudden onset of severe pain, extending along the whole spine, greatly increased by movement, and radiating into other parts, due to irritation of spinal nerve-roots. There is also more or less rigidity of the back from pain and muscular spasm. Paralysis and anæsthesia, up to the level of the lesion, soon follow; but both are incomplete, and do not last long, while anæsthesia may be altogether absent. Recovery is not infrequent.

ACUTE MYELITIS.

Although the connotation of acute myelitis has become greatly restricted, as the result of better knowledge of the subject, yet it still includes conditions which cannot be regarded as uniformly of inflammatory nature, as the title would seem to imply. Perhaps it may be best described and understood as an acute affection of the cord, commonly focal but sometimes diffuse, characterised by rapid disorganisation of the constituent elements of nervous tissue—in other words, by acute softening of the cord. The pathological process varies in different cases. In one group it is of an inflammatory nature, induced by infection of some kind; in a second, it is degenerative, induced by toxic conditions; while in a third, it is a necrotic process induced by thrombosis—chiefly in connection with syphilis. The

histological appearances vary accordingly. The first is characterised mainly by hyperæmia and cell-infiltration; the second by evidences of parenchymatous degeneration, coupled with œdema; and the third by "yellow softening." These three types of myelitis may not be sharply differentiated, and transitional forms are met with.

Etiologically, acute myelitis may follow some specific fever or gonorrhœa; or it may occur in connection with syphilis, pregnancy, or alcoholism. Exposure to wet and cold, and severe muscular exertion may be ranked as predisposing causes. Often, however, no obvious cause can be discovered.

SYMPTOMS.—The most characteristic yet relatively uncommon type is *general* or *diffuse myelitis*, which, from the fact that it generally begins in the lower cord and spreads upwards, is often called *ascending myelitis*. It develops rapidly, with chilliness and high fever followed by pain in the back, increasing numbness and weakness of the legs, until all sensation and power of motion in these parts are lost. Both deep and superficial reflexes are abolished below the upper limit of the lesions, and the bladder and rectum are unable to retain their contents, which pass away involuntarily. Very acute cases may end fatally in the course of a week or two.

In *transverse myelitis*, where only a limited part of the cord is affected, the initial symptoms are less violent, and the progress less rapid. The *dorsal* region is most often affected. There may be some pain in the back and limbs at the outset, but pain is not a common or prominent feature of myelitis. Weakness of the legs is usually first complained of, which increases somewhat rapidly until complete spastic paraplegia is established of all muscles below the level of the lesion. Meanwhile anæsthesia develops over the same area, usually beginning in the feet, and extending upwards, becoming more and more complete.

The knee-jerks are increased, ankle-clonus is readily elicited, and the plantar reflex is extensor. The muscles do not waste, and their electrical reactions are normal. There is usually some retention of urine at first, followed by intermittent incontinence. When the lesion is *cervical*, the symptoms are very similar, but the muscles of the thorax and arm are also involved.

As soon, however, as the physiological continuity of the cord is *completely* interrupted by the lesion, *whatever its level*, the picture changes. Spasticity disappears, the affected muscles rapidly become flaccid and waste, the knee-jerks are lost, and the skin-reflexes (below the lesion) steadily decline—the plantar reflex being the last to disappear. The sphincters lose their tone, and the urine dribbles away.

Recovery may take place occasionally, even after *complete* transverse lesions. In the latter case, there is a reverse change from the flaccid to the spastic state, the first signs of which are the return of the knee-jerks, and extensor plantar reflex. Recovery may be checked in the spastic stage, or be completed. More often, however, death occurs from some complication such as cystitis, bed-sore, pulmonary affections, or kidney disease. Cases of the ascending type usually go from bad to worse, and death may occur early.

The symptoms of compression of the cord resemble those of transverse myelitis in many respects, but the development of symptoms is generally much slower; and pains from irritation of the roots of spinal nerves are a marked feature. Again, the majority of cases of compression are associated with spinal caries. In spinal hæmorrhage the onset is more sudden; and in hysterical paraplegia, the knee-jerks are seldom lost, the plantar reflexes are never extensor, there is no incontinence of urine, and no reaction of degeneration in the affected muscles.

COMPRESSION OF THE CORD.

Among the causes of compression of the cord, spinal caries and tumours are the most important, the former being the more common.

Spinal caries is most frequent in children and young adults. It is due to tuberculous disease, which generally begins in the body of the vertebra, and extends backwards into the spinal canal, where it gives rise to fungoid granulations and thickening of the spinal membranes, which spread upwards and downwards for some little distance. In some cases, suppuration takes place, forming a local abscess. As the disease proceeds, local deformity of the spine commonly occurs—usually a kyphosis—from the collapse of one or more vertebral bodies. It will thus be seen that compression may occur either from thickened membranes, an abscess, or displacement of bone. Apart from compression, the cord may also be injured in spinal caries by persistent local congestion and œdema, due to strangulation of veins by the fungoid granulations; or (more rarely) a secondary local tuberculous meningo-myelitis may develop.

The *symptoms* of spinal caries may be grouped according as they relate to the bony spine, the nerve-roots, or the spinal cord. The earliest symptom is a dull aching pain, confined to the affected part of the spine—more commonly in the dorsal region, increased by movement of any kind, and by deep pressure. This is followed by angular or rounded deformity of the spine. Nerve-root symptoms are characterised by sharp pains radiating into the area supplied by the affected root, accompanied by hyperæsthesia, or a feeling of tightness round the trunk (girdle-pain); and later on by anæsthesia. The symptoms of compression of the cord are, in the main, those of transverse myelitis;

but the onset is much slower as a rule, and evidence of root-irritation is more common.

The course is very chronic. More often, symptoms of compression occur about the same time as marked deformity of the spine, but sometimes a long period intervenes between the appearance of deformity and the occurrence of compression, or the latter may not occur at all. In compression of the cord from tumour, the symptoms are the same as those of caries in the main, with the exception of spinal deformity.

ACUTE ANTERIOR POLIO-MYELITIS.

Acute anterior polio-myelitis is an acute form of paralysis, chiefly met with in young children, especially during the second year of life (*infantile paralysis*), but adults are sometimes affected. Its causes are obscure. An infective origin is suspected, but has not yet been proved. It appears sometimes as a sequel of an acute specific fever, exposure to cold, or a fall; and sometimes it occurs in an epidemic form. A large majority of all cases occur in the autumn quarter of the year.

The lesion on which it depends is an acute inflammation of the anterior cornua of the cord, causing destruction of one or more groups of cell-bodies belonging to the lower motor neurons, and consequent degeneration of their axons in the anterior spinal roots and spinal nerves, together with atrophy and degeneration of the muscles in connection with them. This is followed by fibroid transformation and shrinkage of the cornua.

SYMPTOMS.—The onset is rapid, and marked by fever, which may either be considerable and continue for some days, or be so slight as not to attract attention. In some cases the constitutional disturbance may be so marked that the paralysis escapes observation until the child is well

enough to leave his bed. The paralysis develops quickly, usually reaching its maximum in a day or two, though sometimes later. It may involve a single group of muscles, the whole of one limb, one leg and one arm (of the same or opposite sides), both arms or legs, or all four limbs at once. With the onset of paralysis, there is evidence of considerable pain. The child resents handling, and cries on any movement of the affected limbs. This gradually passes off in the course of a week or ten days as a rule. Otherwise sensation is not affected, neither are the functions of the bladder or rectum disturbed. After remaining stationary for a variable time, the paralysis begins to pass off. Complete recovery is rare; and more often improvement is incomplete, leaving a special group of muscles, or the whole of one limb, permanently paralysed. Those muscles which are not destined to recover begin to waste and exhibit the reaction of degeneration some three or four weeks from the onset. In these muscles the wasting increases, the temperature of the part falls; the development of the limb is more or less arrested; and permanent deformities may arise from altered tension of the muscles upon the joints. It is rarely fatal except when the thoracic muscles are involved.

An exactly analogous condition may affect the grey matter of the medulla, causing an acute bulbar paralysis, but it is rare.

SYSTEMIC NERVOUS DEGENERATIONS.

Many nervous diseases are characterised by degenerative changes which are more or less limited to special physiological tracts of nerve-fibres, and are accordingly termed system-degenerations. Many of the best-known forms of system-degeneration are most easily traced in the spinal cord; but we now know that such degeneration or sclerosis

frequently depends upon diseases of remote nerve-cell-bodies with which the degenerated tracts are structurally connected. In some forms, especially those of a very chronic type, the isolation of particular nervous tracts is remarkably distinct; while in others, although one tract is mainly affected, other parts are also involved to a variable extent. The following classification of the more important system-degenerations is suggested, based upon their distribution:—

SCLEROSIS.	Afferent sensory tracts		. . .	Tabes dorsalis (locomotor ataxy).
	Efferent motor tracts	{	Upper neurons.	{ Cerebral diplegia.
			Lower neurons.	{ Primary spastic paraplegia.
			Both upper and lower neurons.	{ Progressive muscular atrophy.
	Mixed afferent and efferent tracts	{	. . .	{ Bulbar paralysis.
				{ Amyotrophic lat. sclerosis.
				{ Friedreich's disease.
				{ Hereditary cerebellar ataxy.
				{ Sub-acute combined degeneration.

ETIOLOGY.—Strain and stress, mental or physical excesses and alcoholism are often advanced as causes of sclerosis, but are probably only contributory. Tabes more often occurs in those who at some previous time have contracted syphilis; but it should be remembered that only a small proportion of syphilitic subjects subsequently develop nervous degeneration, and that we are ignorant of its determining factor. In other cases degeneration seems to arise from insufficient vitality of the particular tracts or neurons (*abiotrophy*), and may be inherited, or occur in several members of the same family (*familial*).

I.—Degeneration of Afferent Sensory Tracts.

TABES DORSALIS—LOCOMOTOR ATAXY.

In this disease we have to do with chronic degeneration of the posterior-root-fibres, chiefly after their entry into the posterior columns of the cord. The precise distribution of the lesion varies, but it usually begins in the sacral and lumbar regions, with an ascending degeneration extending into the postero-median columns of Goll. In severe cases

nearly the whole thickness of the posterior columns may be involved. Sometimes, but much more rarely, the process begins in the cervical region of the cord. The degeneration is at first very selective and it is unusual to find complete degeneration of the whole of any particular root in the early stages of the disease. In addition to the spinal lesions, analogous changes may take place in the optic and other cranial nerves.

Antecedent syphilitic infection is by far the most frequent, if not the exclusive cause of tabes, with physical and mental overstrain as the most important contributory causes. It is most common in men of middle age.

Symptoms.—In the early or so-called *pre-ataxic stage* the symptoms vary very much in different cases; so much so that a precise consecutive account for general application is impossible. One of the commonest and earliest symptoms consists of sharp "*lightning pains*" down the limbs, due to sensory nerve-root irritation. Loss of knee-jerks, and loss of the reaction of the pupils to light whilst the reaction to accommodation is preserved (Argyll-Robertson pupil) are also important early symptoms. These three symptoms, taken together, suffice for an almost certain diagnosis of tabes.

Among other symptoms of the pre-ataxic stage, the following require notice. *Sensory disturbances* in the form of numbness and formication in the extremities, especially in the soles of the feet, yielding a sensation as of treading on soft carpet; sensation of tightness round the waist (girdle-pain): patches of diminished tactile sensibility; and marked delay in the transmission of pain-impressions are often observed. *Visual disturbances* are also common. The Argyll-Robertson pupil has already been referred to. In addition, the oculo-motor nerves—especially the third and sixth—are often paralysed, giving rise to ptosis of the upper eyelid and squint. Primary atrophy of the optic

disc is present in a certain proportion of cases, and is sometimes a very early symptom preceding ataxy for many years. It progresses very slowly, with failing vision, and ultimate blindness. Disturbance of other spinal senses is less frequent. Another rather characteristic condition is *loss of tone in the muscles* of the affected limbs, thus admitting a much wider range of *passive* movement than normal. *Visceral crises* are not at all uncommon; and, often occurring quite early, their true significance is apt to be overlooked. Gastric crises are the most common, with sudden attacks of gastric pain and vomiting. In other cases the crisis involves the bowel, causing sharp attacks of abdominal pain and diarrhœa; or the larynx, causing urgent attacks of dyspnœa. *Vesical disturbance* is frequently met with, in the form of painful or frequent micturition, or slight delay in starting the act. *Constipation* is the rule owing to loss of power in the colon. Sometimes *impotence* is an early symptom, though occasionally there is early sexual excitement.

The second stage of tabes is characterised by a *gradually increasing muscular inco-ordination*, mainly due to progressive loss of muscular sense. In the first instance it does not amount to more than uncertainty of gait, especially when walking in the dark, or turning sharply. In course of time true ataxy develops (*ataxic stage*), and the patient is unable to stand with his feet together and his eyes shut (*Romberg's sign*), while the gait is characteristic. The act of walking requires the closest watchfulness on the part of the patient, the leg which is in movement is thrown irregularly forward with unnecessary violence, and is brought to the ground—heel first—with unnecessary force, until at last his movements become so uncertain and tumultuous that walking is impossible. But with all this, there is no marked loss of muscular power; the muscles for a long time maintain their nutrition and normal electrical

reactions; and, so long as the patient can walk at all, he does not become fatigued very readily. The inco-ordination may extend into the arms, manifested by an awkwardness in using the fingers for buttoning the clothes, or picking up a small object. There is also a marked difficulty in accurately controlling the hands for any delicate movement, as in writing. "Lightning pains" generally diminish or disappear as the disease advances.

Various *trophic lesions* may also occur, of which the following are the chief:—temporary œdema, herpes, perforating ulceration of the foot, special brittleness of the bones leading to so-called "spontaneous fractures," and certain joint-changes. These last (*Charcot-joint*) are so remarkable as to demand a short description. Any joint of the affected limbs may suffer, but the knee most commonly. It begins suddenly with much swelling of the joint, and its immediate neighbourhood. There is no pain, tenderness, or heat of the part. After a variable interval the swelling subsides to a greater or less extent; but the cartilage meanwhile becomes eroded, the bony structures waste and become disorganised, and the ligaments become relaxed, so that the joints admit of an abnormal range of movement. Periarticular thickening and bony excrescences are apt to form, and the muscles around the joint undergo atrophy. In spite of this, it is remarkable how long the patient manages to get about.

Locomotor ataxy, though occasionally acute, generally runs an extremely tedious course, extending over many years. A single symptom may exist alone for a long time, but sooner or later others appear, and gradually tend to become worse and worse. In the last, or *paralytic stage*, the patient wastes and becomes unable to walk, and in this weak and helpless condition, the anæsthesia of the affected limbs may become more or less complete, bed-sores may develop, the urine and fæces may dribble away, and the

patient may finally die from sheer exhaustion. More often, however, death occurs from some intercurrent trouble, such as renal disease or general paralysis of the insane.

II.—Degeneration of Upper Motor Neurons.

All degenerations affecting the integrity of upper motor neurons give rise to paralysis of a spastic type; and when the legs only are involved, we speak of the condition as one of *spastic paraplegia*. It is characterised (as are all spastic paralyses) by muscular weakness, rigidity, increased deep reflexes, and plantar reflex of the extensor type. In early childhood it is not an uncommon result of a cerebral lesion due to meningeal hæmorrhage, congenital defect, hereditary syphilis, or certain inflammatory conditions. In such cases it may be associated with spastic paralysis of the upper limbs as well, and under these circumstances it is generally described as *diplegia*. The symptoms of cerebral diplegia in infants have already been considered under the head of hæmorrhage into the cerebral membranes (p. 525). More often, however, spastic paraplegia is due to lesions in the spinal cord. As a rule, it occurs either as a direct secondary result of other spinal diseases, as compression of the cord, transverse myelitis, or hæmorrhage: or in association with other symptoms, as in amyotrophic lateral sclerosis, some cases of disseminated sclerosis, and syringomyelia. A primary form of spastic paraplegia is described, but it is rare.

PRIMARY SPASTIC PARAPLEGIA.

The patient first complains of weakness, premature fatigue, and more or less stiffness on walking, and one leg is generally affected a considerable time before the other. The difficulty slowly increases, until a definite *spastic gait* is established, presenting the following features. Movement is hindered, the steps are short, and the legs tend to

move as a whole. The feet seem to cling to the ground ; and, in order to assist forward movement, the pelvis is raised on the same side, and the leg is swung round. Owing to adductor spasm, there is a tendency for the moving leg to cross the other as it comes forward. There is no disturbance of sensation, and the muscles preserve their nutrition and normal electrical reaction. The reflexes are exaggerated, ankle-clonus is readily elicited, and the plantar reflex is extensor. Sometimes the arms are also involved. The course is very slow, extending over many years, and death is more likely to occur from intercurrent disease.

A form of *hereditary spastic paraplegia* is occasionally met with. It begins in childhood or adolescence, affecting boys more often than girls. Several members of the same family may be attacked.

III.—Degenerations of Lower Motor Neurons.

PROGRESSIVE MUSCULAR ATROPHY.

Progressive muscular atrophy is due to a chronic progressive atrophy and degeneration of the nerve-cells in the anterior cornua, *without* implication of the pyramidal tracts. Thus restricted, it is no doubt a rare disease. Most of the cases hitherto classed under this head should probably be described as amyotrophic lateral sclerosis. Its etiology is obscure. It is a disease of middle life, more common in males than females.

The onset is insidious. Attention is usually first attracted by weakness and wasting of the muscles of the ball of one thumb, so that it cannot be firmly opposed to the forefinger, or abducted. The atrophy and weakness gradually increase, and other muscles tend to become involved, for the most part in the following order :—the lumbrical and interosseal muscles, so that the metacarpal bones stand out more distinctly ; the small muscles of the little finger ;

the muscles of the forearm, and some of those of the arm, together with the deltoid and shoulder muscles. The triceps, latissimus dorsi, and the lower part of the great pectoral more often escape. As a result of the interosseal paralysis, a claw-like deformity of the hand occurs, characterised by extension of the metacarpo-phalangeal joints, and flexion of the phalangeal joints (*main en griffe*).

At a later stage, the extensors of the neck may be affected, so that the head bends forward; or (less commonly) the legs may be involved. Sometimes the trouble begins in the shoulder muscles instead of the thumbs. But wherever it begins, the corresponding muscles on the opposite side are the next to suffer, and the disease advances almost simultaneously on the two sides of the body. *Fibrillary tremors* in the affected muscles are of common occurrence or may be readily evoked by sharply tapping them. Loss of power only occurs as the result of and in direct proportion to the amount of wasting. The electrical reaction of degeneration develops very gradually, and only becomes typical when the atrophy has involved the bulk of the affected muscles.

The course is very slow, usually extending over several years, with stationary periods—often of long duration. Death ultimately takes place from implication of the respiratory muscles, from exhaustion, or from some complication.

CHRONIC BULBAR PARALYSIS.

This disease is characterised by a paralysis of the tongue, lips, soft palate, and pharynx, which depends upon degeneration of the motor nuclei in the medulla. In many cases, if not in most, degeneration also involves the adjacent motor tract; and thus some writers have described it as a form of amyotrophic lateral sclerosis.

Symptoms.—The onset is gradual. The first symptom

is usually a difficulty in articulation, due to a want of precision in the movements of the tip of the tongue. The latter gradually wastes and weakens, and exhibits fibrillary tremors, until at last it can be no longer protruded, but lies motionless, or nearly so, in the mouth. Meanwhile the paralysis and wasting slowly involve other parts. The *soft palate* hangs loosely in the throat, and therefore the articulation of explosive consonants is difficult. Owing to *weakness of the hyoid group of muscles*, the early stage of deglutition is impeded, and food is liable to pass into the glottis or nose. The muscles of the larynx also become weakened. Curiously enough, while the orbicularis oris becomes paralysed, so that the lips cannot be kept together, the other facial muscles are not affected. As the symptoms get worse, the condition of the patient is pitiable indeed. Articulate speech becomes impossible, though phonation is not entirely lost; the saliva cannot be properly swallowed, and on accumulating dribbles from the open mouth; food collects in the sides of the mouth, can only be swallowed with great difficulty, and is apt (especially if liquid) to enter the larynx or to return through the nose. The same imperfect and slowly developing reaction of degeneration in the affected muscles is observed as in progressive muscular atrophy, owing to the mixture of relatively healthy and degenerate fibres in the same muscles. There is marked emotional excitability, the patient often crying and laughing at the same time. If the motor tract is also implicated, the tendon reflexes of the limbs are increased, and a marked lower jaw-jerk may be present.

The disease is always fatal, with an average duration of about two years. Death either occurs from starvation, asphyxia, syncope, or some intercurrent disease such as broncho-pneumonia.

IV.—Degeneration of both Upper and Lower Motor Neurons.

AMYOTROPHIC LATERAL SCLEROSIS.

The degenerations of lower motor neurons just described are often associated with corresponding degeneration of the upper neurons, and such a combination is known as amyotrophic lateral sclerosis. It is characterised therefore by spastic paralysis of some parts, and atrophic paralysis of others. The distribution of each, and the order of their development vary considerably in different cases. Thus some cases begin with progressive muscular atrophy, or bulbar paralysis, and subsequently develop spastic signs; while in others the primary condition is one of spastic paraplegia, associated later on with atrophic changes. In fact, the disease represents a more or less diffuse degeneration of motor neurons. The prognosis is very serious, and the average duration is from one to three years.

V.—Combined Degeneration of Sensory and Motor Neurons.

FRIEDREICH'S HEREDITARY ATAXY.

This is a somewhat rare disease, characterised by progressive degeneration of the posterior and lateral columns of the cord. It tends to occur in successive members of the same family, but is not directly inherited. The cause is unknown. It usually comes on in childhood from five to sixteen years of age. It is characterised by slowly advancing ataxy, until the patient becomes bed-ridden. Other symptoms, such as slurring speech, nystagmus, scoliosis, and clubbing of the feet are not uncommon. The knee-jerks are lost, but the skin and pupillary reflexes are usually normal. The course of the disease is very chronic, and death usually occurs from intercurrent disease.

HEREDITARY CEREBELLAR ATAXY.

This is a still rarer condition of the same type, in which the cerebellar tracts are chiefly affected. The symptoms are much the same as in Friedreich's disease, but the knee-jerks are normal or exaggerated. Foot-deformity is absent, and begins at somewhat later age.

SUB-ACUTE COMBINED DEGENERATION.

This is a progressive disease, usually attacking the badly nourished of either sex in middle life, and is characterised by spasticity and ataxy. It is almost always associated with some severe toxic condition ; and is characterised anatomically by progressive degeneration, beginning in the posterior columns, and gradually involving the whole thickness of the cord—the early symptoms are those of ataxy, with slight stiffness of the legs, increased knee-jerks, and extensor plantar reflex. Later, the condition is one of complete spastic paraplegia, with considerable loss of sensation ; while finally, complete flaccid paralysis supervenes, with incontinence of urine and fæces, and bed-sores, until death takes place from asthenia, usually within two years from the onset.

TUMOURS OF THE CORD AND MEMBRANES.

Under this head are included growths in the substance of the cord, growths in the membranes, and growths in the vertebræ intruding into the spinal canal. The list of possible growths is a long one, but by far the larger number occur in connection with cancer, tubercle, or syphilis, and affect the spinal cord by encroaching upon it from without inwards. The symptoms to which such growths give rise may be classed in two groups :—(a) those due to irritation of the membranes, cord, and nerves

arising from it; and (b) those due to compression of the cord.

Symptoms of irritation.—The duration of the stage of irritation varies greatly in different cases—from a few months to two or three years. The most prominent symptom is pain in the back at the seat of the growth, which radiates into the area supplied by the irritated root; at first neuralgic and paroxysmal, but tending later to become duller, aching, and more constant. It may at first be unilateral, but more often becomes bilateral at a later stage. In the trunk it may be associated with girdle-pain. Trophic troubles may also occur, such as herpetic eruptions, or sloughing sores.

Symptoms of compression.—These have already been described on page 541. Intra-medullary tumours are less likely to be attended with pain; and the symptoms are usually those of a gradually progressive paraplegia and anæsthesia. The symptoms are more often bilateral from the first; and muscular atrophy appears earlier and is more marked and extensive.

SYRINGOMYELIA.

Syringomyelia is characterised by cavities in the cord, either as the result of developmental defect, or degeneration of neuroglial overgrowth. The cavities may or may not communicate with the central canal. The cervico-dorsal region of the cord is most often affected. Although most cavities are probably of congenital origin, yet the possibility of formation in later life must not be ignored. In any case, however, symptoms do not usually appear until early adult life.

Three groups of symptoms should be noted: (1) An atrophic paralysis, closely corresponding to progressive muscular atrophy, generally affecting the muscles of the

hands, arms, and shoulder-girdle; (2) a curious dissociation of sensibility, marked by failure to appreciate impressions of heat or cold, but with the retention of tactile and muscular sensibility; and (3) trophic disturbances affecting the skin, nails, and joints. The lower limbs are more or less spastic, with increased knee-jerks, and extensor plantar reflexes. In the comparatively rare cases, where the lesion affects the lower part of the cord, the above symptoms would be correspondingly modified. Its progress is very chronic, and death ultimately occurs either from hæmorrhage into the cord, or intercurrent disease.

MUSCULAR DYSTROPHIES.

Three types of muscular dystrophy will be described:—the juvenile form (Erb), the facio-scapulo-humeral form (Landouzy-Déjerine), and pseudo-hypertrophic paralysis. They depend upon *primary changes in the muscles*, as contrasted with atrophy arising from neural lesions, central or peripheral. For the most part, they are confined to childhood, are apt to affect successive members of the same family, and may be inherited. Their cause is unknown.

Anatomically, the muscles at first hypertrophy, but afterwards waste, with marked increase in the connective tissue. Fat may be deposited in the latter to such an extent that the muscles appear to be large, though actually much atrophied (*pseudo-hypertrophy*). There is no change in the central nervous system.

THE JUVENILE FORM usually begins somewhere between puberty and twenty years of age. It most frequently affects the muscles of the arm and shoulders, often extending later to the muscles of the trunk. The forearms and hands are rarely affected. Sometimes the muscles of the

pelvic girdle are first affected. The only symptoms are wasting and weakness. There is no sensory disturbance, no fibrillary tremor, no reaction of degeneration, and no affection of the sphincters. The wasting progresses up to a certain point, and then generally becomes stationary and remains so to the end of life.

THE FACIO-SCAPULO-HUMERAL FORM is rarer than the juvenile, and begins in quite early childhood with weakness of the orbicularis muscles of the eyelids and mouth, so that the eyes cannot be properly closed, nor can the lips be pursed up. Other muscles of the face may be involved, causing a curious change of expression. Later on, the muscles of the shoulder-girdle become affected, as in the previous form.

PSEUDO-HYPERTROPHIC PARALYSIS is most common in boys from two to six years old. The muscles of the calves are most frequently affected, but subsequently those of the thighs or arms may become involved. The earliest symptoms consist of weakness of the legs, and a peculiar straddling waddling gait. The shoulders are thrown back, and the belly is thrust forwards. The affected muscles are soon found to be unduly large and prominent; and as the enlargement increases the weakness becomes more marked, until at last both walking and standing are impossible. The child cannot stoop to pick anything up from the floor; and the laborious process of raising himself from a prone to the erect posture by lifting his body up with his hands on the legs is very characteristic. Associated with these enlarged muscles, are others which are only wasted; and in the later stages, the enlarged muscles also diminish in size. Deformities, such as talipes equinus, may then occur. The course of the disease is slow, the average duration being about five or six years. Death takes place from exhaustion, implication of the respiratory muscles, or some intercurrent disease.

MYASTHENIA GRAVIS.

This is a rare affection of young adults, characterised by extreme fatigue on slight exertion and muscular weakness, without any discoverable neuro-muscular lesion, generally beginning in muscles supplied by cranial nerves, but subsequently extending to all the muscles of the body. The weakness is increased by exertion, but is recovered by rest. The muscles do not waste, and the only peculiarity about their electric behaviour is that while they respond normally to Faradism at first, they soon become exhausted and fail to contract (myasthenic reaction). Temporary recovery may take place, but with a tendency to subsequent relapse ; and the majority of all cases end fatally.

GENERAL DIAGNOSIS OF DISEASES OF SPINAL CORD.

The diagnosis of disease of the spinal cord involves, first of all, determination of its *seat*, and afterwards of its *pathological nature*.

Localisation in a vertical direction is determined (*a*) by noting the distribution of paralysis (when present) in connection with the origin of the nerves by which the affected muscles are supplied : (*b*) by the behaviour of the various reflexes—superficial and deep—in connection with the level at which their corresponding reflex arcs pass through the spinal cord ; and (*c*) by observing the area of any sensory disturbance in relation to the spinal segment with which it is associated.

Localisation in a transverse direction depends upon evidence of interference with the various tracts of white and grey matter, or nerve roots. Thus, destructive lesion of the crossed pyramidal tract is associated with spastic paralyzes ; of the posterior columns, with ataxy ; of the

anterior cornua and anterior nerve-roots, with flaccid atrophic paralyses; of the posterior cornua, with loss of sensation to pain and temperature, while tactile sensation may be normal; and of the posterior nerve-roots, with anæsthesia.

With regard to the pathological nature of diseases of the cord, the following considerations may be of use; and, first of all, in regard to mode of onset.

A *sudden onset* always points to a vascular lesion—almost always to hæmorrhage. The chief diseases with an *acute* or *rapid onset* are acute meningitis, myelitis, and anterior polio-myelitis. Pain and spasm, with Kernig's sign, are the most characteristic features of *acute meningitis*, which rarely occurs except in association with cerebral meningitis, or as secondary to local injury or disease of the spinal column. In doubtful cases, lumbar puncture may be of great assistance. In *acute ascending myelitis* pain is not prominent, and paralysis with anæsthesia rapidly develop. The sphincters are paralysed, and bed-sores appear early. *Acute anterior polio-myelitis* is mainly a disease of young children; there is no anæsthesia, and the sphincters are not implicated.

Chronic cases with *gradual* onset fall into three main groups, according as the dominant symptoms are those of spastic paralysis, of flaccid atrophic paralysis, or of muscular inco-ordination or ataxy. With the exception of primary lateral sclerosis, which is a rare disease, spasticity is seldom met with apart from other symptoms. The principal diseases in which *spastic paralysis* is met with are:—transverse myelitis, compression of the cord, syringomyelia, sub-acute combined degeneration, and amyotrophic lateral sclerosis. Disseminated sclerosis (p. 513) should also be remembered. The diagnosis of transverse myelitis has already been given; and besides, its onset is rapid. In *compression of the cord*, spasticity is associated with

local pain and tenderness over the spine, with radiating pain at the level of the lesion, followed after a variable interval by paralysis and anæsthesia below the lesion. As between compression due to caries, and that due to tumour—when deformity of the spine is present, diagnosis is easy. In tumour, the symptoms are often unilateral at the onset, the progress is slower, and anæsthesia may be delayed. In *syringomyelia*, spasticity is accompanied with progressive atrophy and dissociated sensation in the arms. In *sub-acute combined degeneration*, spasticity is associated with ataxy in the early stages, and the condition ultimately merges into one of flaccid paraplegia. In *amyotrophic lateral sclerosis*, spasticity is commonly preceded by atrophic paralysis of the muscles of the hand and arm.

The principal diseases of the cord in which *muscular atrophy and wasting* are marked features, are:—acute anterior polio-myelitis (distinguished by its acute and rapid onset), progressive muscular atrophy, progressive bulbar palsy (scarcely to be mistaken), amyotrophic lateral sclerosis, and syringomyelia. *Progressive muscular atrophy* is distinguished from amyotrophic lateral sclerosis, by the absence of spastic signs; and from syringomyelia, by the absence of dissociated sensory symptoms. The wasting of *muscular dystrophies* may be distinguished by the early age at which the latter occur, their hereditary and familial tendencies, affection of proximal rather than distal parts of a limb, absence of fibrillary tremors, and absence of reaction of degeneration. In *multiple neuritis*, muscular wasting is bilaterally symmetrical, develops somewhat rapidly, and is associated with sensory disturbances and tenderness of the muscles.

Tabes dorsalis is the commonest and most important *ataxic* disease of the spinal cord. Friedreich's disease may be distinguished from it, by its occurrence in childhood, normal sensation, normal pupils and skin-reflexes,

nystagmus, disturbance of speech, and club-foot. The ataxy of *combined degeneration* is associated with increased reflexes, absence of the Argyll-Robertson pupil, and definite spasticity at some stage of the disease. In ataxy from *cerebellar disease*, the reeling gait, vertigo, vomiting, and optic neuritis are sufficiently characteristic. Ataxy occurring in association with *multiple neuritis* may sometimes give difficulty; but the more rapid onset, evident loss of power, dropped feet, muscular tenderness, reaction of degeneration, and the history, usually suffice to distinguish it.

It is an all-important fact that *hysteria may simulate almost every organic nervous disease*, and great care is needed to distinguish such cases. There are certain signs which occur in organic disease which never occur in hysteria—namely, optic neuritis, optic atrophy, squint and diplopia, incontinence of urine and fæces, and extensor plantar reflex. On the other hand, it must be remembered that retention of urine may be hysterical, and that knee-jerks may be greatly increased and ankle-clonus may be present in purely functional conditions. The sensory disturbances of hysteria are fairly characteristic, but not absolutely so. In the limbs anæsthesia affects areas, the upper limits of which are approximately described by a circle round the limb at some point, as distinguished from the areas of anæsthesia in spinal cord disease, which usually run parallel to the axis of the limbs, or nearly so. Disseminated sclerosis may be simulated by hysteria with especial closeness, but there is no change in the fundus oculi, no nystagmus, and no squint; and though tremor may be present, it is not of the typical intention type. A peripheral contraction of the field of vision is characteristic of hysteria, whereas in disseminated sclerosis, if the field of vision is curtailed, it is in the form of a central scotoma. Aphonia indicates hysteria, but does not negative organic disease.

Muscular weakness in hysteria is often much more marked on attempting some common action such as walking, than in moving the legs in bed (abasia). In organic disease no such difference is observed. Though the reflexes are often supposed to be unaltered in hysteria, only the corneal, pupillary, and plantar reflexes can be relied upon. The last is of special importance, since it is always extensor in all upper motor neuron disease, while in hysteria it is often absent, sometimes flexor, but never extensor.

GENERAL TREATMENT OF CENTRAL NERVOUS DISORDERS.

In *vascular lesions of the brain* during the apoplectic attack, the patient must be kept perfectly quiet in the recumbent posture, with the head slightly raised, and all clothing should be loosened, especially about the neck. Ice should be applied to the head, but the body should be kept warm. Five grains of calomel may be mixed with a little butter, and placed well back on the tongue. If the breathing be obstructed or stertorous, the patient should be turned on his side. No food is necessary for the first day, but if the patient is then unable to take nourishment by the mouth, nutrient enemata should be resorted to. When the pulse-tension is high, inhalation of nitrite of amyl may be used from time to time; but no medicines are needed except in syphilitic cases, when the sooner a course of iodide of potassium and mercurial inunction can be adopted the better.

In *acute inflammation of the brain and membranes*, our chief therapeutic resources are of an indirect kind. The patient's room should be cool, well ventilated, and quiet; the hair should be cut short, or (in severe cases) shaved to the scalp, followed by the application of cold in the

form of an ice-cap or cold-water coil, or by the application of a few leeches to the temples or behind the ears if there be much pain and irritability. The bowels should be actively opened from time to time by a brisk aperient, such as calomel and jalap—gr. v. of each for an adult, and $\frac{1}{4}$ to $\frac{1}{2}$ that dose for a child. As general calmatives in addition to the cold applications to the head already mentioned, the most useful are—the tepid wet sheet pack, mustard pediluvia, or the wrapping round the feet and legs with cloths wrung out in strong mustard and water, bromide of potassium, chloral hydrate, and morphia. The latter should be used cautiously, but the addition of a small quantity to a bromide or chloral draught at bed-time is often advantageous, especially if there is much pain. When opiates are contra-indicated, as with young children, tincture of henbane may be substituted. The diet should be bland, plain, and nutritious; and if there be any difficulty about feeding by the mouth, nutrient enemata must be persevered with. Alcohol is not desirable as a rule; but in the later stages where there is marked prostration it may be needed.

Acute inflammation of the cord and its membranes requires analogous treatment. Dry cupping alongside the spine affords a further measure of some value, in addition to those already mentioned, for the purpose of reducing local congestion. In all acute central nervous affections the bladder should be periodically examined with care, especially in affections of the cord, and if there be retention, the urine should at once be drawn off. If the bladder be inflamed, it should be frequently washed out with some anti-putrefactive solution, such as a warm solution of boracic acid.

The treatment of *chronic cerebral and spinal disease* is far from satisfactory. Physiological rest of the nervous system should be aimed at; and the general health must be

supported by good food, fresh air, and the use of tonics and cod-liver oil. In many cases the internal free administration of iodide of potassium and mercurials is most beneficial, especially in those of a syphilitic nature. Phosphorus, phosphide of zinc, Donovan's solution, and nitrate or oxide of silver are also serviceable nervine tonics in chronic degenerative diseases of the central nervous system.

The present is also a convenient place for referring to the leading principles of the treatment of the more important symptomatic states referable to nervous disturbance.

Headache.—Careful search for likely causes must be made, *e.g.*, carious teeth, visceral disturbances, exhaustion, gastrointestinal trouble (especially constipation), rheumatism, gout, syphilis, and organic cerebral disease; and endeavours should be made for their correction or removal. Too much stress cannot be laid upon the importance of good hygiene in all classes of cases. Since the toxic effects of imperfectly metabolised products are not only a common primary cause of headache, but a frequent factor in combination with other causes, a light simple dietary, and moderately free elimination from the bowels and kidneys, are generally beneficial. Active purgation, however, should be avoided in the weak, feeble, or aged. The following hints may be of service. In anæmia—ferruginous tonics, arsenic, strychnia, phosphorus, cannabis indica, and alcohol; in congestion—bromide of potassium, ergot, cold applications to the head, and moderate purgation; in dyspepsia—alkalies before meals, mineral acids after meals, bismuth, nux vomica, vegetable bitters, and alterative aperients; in nervous exhaustion—rest, tonics, phosphorus, valerianate of zinc, and (if sleepless) bromide of potassium, chloral, or morphia at night under strict supervision; in syphilis—iodide of potassium; and in headaches from organic disease—bromides, gelsemium, cold applications

to the head, sinapisms to the back of the neck, or leeches behind the ears.

Vertigo.—Common causes like anæmia, gastro-intestinal derangement, ocular troubles, abuse of alcohol, tea, coffee, or tobacco, should be inquired for and removed. In aural vertigo, full doses of quinine with hydrobromic acid, bromide of potassium, or a hypodermic injection of morphia will often relieve an attack at the time, but they do not prevent its recurrence, or interrupt progressive deafness.

Insomnia.—The chief pathological causes of sleeplessness are extreme exhaustion, fever, congestive conditions of the brain, gouty dyspepsia, and certain toxic agents such as alcohol, tea, coffee, and tobacco. Insomnia from exhaustion often demands prolonged rest and change of air and scene. Mustard pediluvia, or a hot full bath at bedtime are of service. Such patients often sleep fairly well for the first part of the night, but wake up in the early hours, feeling low and depressed and get no more real sleep till morning. In these cases, not only should some light nourishment and a little stimulant be taken before retiring to bed, but it should be repeated on waking—once or oftener as required. As to medicines, opiates should be avoided as far as possible; and bromides, chloral, and paraldehyde are the best hypnotics. A course of nervine tonics is also most beneficial. Insomnia during the early stages of fevers is best combated with cold to the head, cold spongings, and tepid wet sheet packing; and, if accompanied with much excitement—bromides or chloral. During the later stages, a frequent supply of nutritious food, discreet recourse to alcoholic stimulants, and careful attention to the excretory functions are specially indicated. The principles of the treatment of congestive headaches are similar to those already given for active cerebral congestion. Sedative drugs may be necessary, but opiates should only be given with caution. The treatment of

dyspeptic and toxæmic insomnia calls for no special remark in this place.

The treatment of *delirium* will vary according to its cause. When it is due to cerebral inflammation, the measures already mentioned must be adopted; when it results from exhaustion and the skin is cool with a feeble pulse, good food and stimulants are called for, and not infrequently an opiate is of great service unless there be coma, renal disease, or pulmonary congestion. In the "typhoid state" alcohol, nourishing food, and tepid sheet-packs, with careful attention to the excretions, are necessary. In delirium from high fever the cold bath or cold sheet-packs are of most benefit; and lastly, if traceable to reflex irritation, the cause of the latter should be carefully investigated and removed if possible, and opium, chloral, or bromide of potassium should be given internally.

The treatment of *coma* also varies according to its cause. In apoplectic coma the patient should be placed in the recumbent posture with the head high, allowed plenty of fresh air, and the clothing loosened. Venæsection may be of benefit if the circulation be vigorous and the breathing free, but under other circumstances it is more harmful than otherwise. The bowels should be freely opened by placing a few grains of calomel in a little butter on the back of the tongue. If the coma be associated with renal disease, it may be uræmic, and then calls for the treatment already described under that head. Coma from opium-poisoning, etc., requires prompt use of the stomach-pump, followed by the administration of strong coffee, and constant flipping and movement in order to keep the patient roused.

In chronic *paralytic* conditions from central nervous disease local treatment is very useful, such as counter-irritation to the spine, systematic rubbing of the muscles (*massage*), and the various forms of electrification.

SUB-SECTION IV.

DISEASES OF THE NERVES.

MULTIPLE NEURITIS.

Neuritis, or inflammation of a peripheral nerve may be set up by local injury, pressure of bones in fractures and dislocations, or possibly by exposure to cold or damp; but *multiple neuritis* is due to general disturbance, affecting several nerves simultaneously or in rapid succession. It is not a primary inflammation, but a degenerative process, due to the action of some poison or other noxious agent directly on the nerve-fibres. Such toxic agents may be derived from many sources. They may be introduced from without, as alcohol, lead, or arsenic. Again they may result from infective disease, such as diphtheria, typhoid fever, or syphilis: or from metabolic disorder, as in diabetes. Lastly, there are specific forms of multiple neuritis, as in leprosy and beri-beri. It is essentially a disease of adult life. Children are rarely affected, except in connection with diphtheria, or long-continued medication with arsenic.

The *anatomical changes* begin and are most marked in the smaller peripheral branches of the affected nerves. In the early stages the nerve is softened and swollen from œdema; but, later on, it becomes shrunken and hard. Microscopically, the myelin sheaths are broken up and disintegrated, and somewhat later the axis-cylinders become varicose and undergo atrophy. Secondary to the parenchymatous changes, a reactionary hyperplasia of the connective tissue elements takes place. In some cases, degenerative changes may be observed in the posterior spinal roots, and in the anterior cornual cells. But the nerve changes differ from those due to nuclear disease in

their beginning in the distal part of the nerves, in their patchy distribution in the fibres, and in the rarity with which the whole thickness of the nerve is affected. The wasting and degeneration of the muscles is mainly due to the disease of the nerves; but possibly in part to the direct action of the toxin.

Multiple neuritis more commonly results from chronic alcoholism than from any other cause, so that the alcoholic form may be taken as a type of the disease. It affects women more frequently than men, and generally begins gradually with numbness and tingling of the hands or feet, with severe aching pains in the same parts. The symptoms are almost always bilaterally symmetrical, though perhaps worse on one side than the other, and are generally more marked in the legs than the arms. Muscular weakness of the limbs on both sides of the body soon follows, beginning at the distal extremities and gradually extending upwards. Owing to implication of the flexors of the ankles and the extensors of the wrists, "dropped feet" and "dropped wrists" are often observed. In order to prevent the toes catching the ground, the legs are raised abnormally high, producing the so-called *step-page-gait*, which is characteristic. The paralysed muscles are soft and flaccid, quickly atrophy, and exhibit the reaction of degeneration. Tenderness of the affected muscles on pressure—sometimes most acute—is another characteristic feature. The deep reflexes are lost, but the functions of the bladder and rectum are not disturbed. In severe cases, various muscles of the trunk may be affected; or the diaphragm may be involved, causing serious embarrassment of the breathing. In protracted cases, there may be some œdema of the paralysed limbs, with trophic changes in the skin and nails. Occasionally ataxy occurs, resulting from loss of common sensation, which may lead to confusion with *tabes dorsalis*. It should

be added that alcoholic neuritis is usually associated with considerable mental and moral perversion. Memory for recent events is commonly impaired, and the word of the patient cannot be trusted.

The course of multiple neuritis is always tedious. In ordinary cases, after ceasing to take alcohol, the symptoms increase in severity for two or three weeks, then become stationary for a time, after which gradual recovery takes place. Muscular tenderness is one of the most persistent symptoms. Death sometimes occurs, either from implication of the respiratory muscles, or from some complication.

The diagnosis is not difficult as a rule, with careful attention to the signs and symptoms described above. The diseases which are most liable to be confounded with neuritis are progressive muscular atrophy, tabes dorsalis, and ascending myelitis.

The principles of *treatment* are as follows :—The primary cause should be carefully inquired for and removed as far as possible. Locally, soothing applications are indicated during the acute stage ; while chronic neuritis demands passive movements, massage, and electrification. Pain may be relieved by morphia injections. Medicinally, iodide of potassium and mild mercurials are of most service ; and in all cases, attention to the general health is of the greatest importance. Avoidance of contractures is often difficult owing to local tenderness, but much may be done by massage and passive movements, or by the use of mechanical apparatus to prevent their occurrence.

OCULAR AND AUDITORY DEFECTS.

Functional Disorder of Vision.—Temporary *amaurosis*, or blindness, is not uncommon in uræmia, and in toxic impregnation with lead or alcohol. *Amblyopia*, or dimness

of sight, is sometimes observed in hysteria; but it is especially frequent in connection with tobacco-poisoning.

Retinitis, or inflammation of the retina, is a common result of hæmorrhagic extravasations into the retina, in connection with Bright's disease, leukæmia, syphilis, and pernicious anæmia. It is characterised by irregular opacities and pigmentations. Very similar changes may occur as the result of degenerative change, independently of hæmorrhage.

Optic neuritis, or *choked disc* is more often due to increased pressure within the cranial cavity, as from tumour; but it also occurs in association with local inflammatory, or general toxic conditions. It is characterised by congestion and swelling of the discs, with blurring of their outlines. The arteries are small and often obscured, but the veins are engorged. Flame-like hæmorrhages are frequent, but not essential. Vision may be unaffected for a long time. It is almost always bilateral. As suggestive of intra-cranial disease, it is of the greatest importance.

Atrophy of the optic disc is often a sequel of inflammatory mischief, but is sometimes primary, as in *tabes dorsalis* for instance. The disc is pale and excavated, its margin is distinct, and the retinal vessels are small. Vision always suffers.

Hemianopia, or blindness in the corresponding half (right or left) of the field of vision of each eye (*homonymous hemianopia*), results from destructive lesions of the optic tract, or of the occipital lobe of the brain on the opposite side. Injury of the optic commissure generally causes blindness in the outer half of each field of vision (*bi-temporal hemianopia*). Hemianopia, of a purely functional nature, is a common feature of migraine.

Diplopia, or double vision, results from a want of parallelism in the axes of the eyes, or squint. In convergent

squint, each image corresponds to the eye of the same side (*homonymous diplopia*) ; while in the divergent squint, the right image corresponds with the left eye, and *vice versa* (*crossed diplopia*). Squint is never caused by cortical lesions of the brain.

Conjugate Deviations of the Eyes.—The conjugate movements of the eyes are under the control of cortical motor centres of the brain—on the right side for movements to the left ; and on the left side for movements to the right. From this point (say, on the right side) fibres communicate indirectly with the nucleus of the left sixth nerve, from which other fibres extend to the nucleus of the right third nerve to control the right internal rectus muscle. A destructive lesion of the right cortical centre or of the right internal capsule will therefore be attended with paralysis of movement to the left, and thus lead to uncontrolled deviation of both eyes to the right, or *towards* the lesion. A destructive lesion of the nucleus, say, of the right sixth nerve, will be similarly attended with conjugate deviation of the eyes, but in this case it will be to the left, or *away from* the lesion.

Variations in the Pupils.—*Myosis*, or persistent contraction of the pupil, may be due to hypermetropia, paralysis of the sympathetic in the neck, photophobia, hæmorrhage into the pons, or certain poisons such as opium. It is common in locomotor ataxy from interference with the cilio-spinal centre in the cervical part of the cord. Dilatation of the pupil, or *mydriasis*, is observed in paralysis of the third nerve, in general debility, anæmia, cerebral compression, and from certain poisons as atropine or cocaine. *Inequality* of the pupils is common in paralysis of the insane, and aneurism of the aortic arch.

Nystagmus is characterised by oscillations of the eyeballs from side to side, usually affecting both eyes symmetrically. It occurs in connection with albinism, cataract, cerebellar

tumours, hereditary ataxy, and disseminated sclerosis. Miners are also especially liable to it, from the persistent strain of looking upward in dull light.

Deafness may be due to a defect in the auditory nerve, or to local disease interfering with the conduction of sound through the ear. Nervous deafness may be distinguished from deafness due to faulty conduction, by means of a tuning-fork made to vibrate in contact with the bones of the head. In the former it cannot be heard; while in the latter the sound is intensified. Nervous deafness may occur in fractures of the base of the skull, Menière's disease, *tabes dorsalis*, and senility.

Tinnitus aurium is characterised by subjective sensations of ringing, rushing, or humming noises in the ear. It may be caused by any local ear-disease, or general disorder which irritates the auditory nerve, and is often a most intractable symptom. It may be associated with deafness and vertigo.

LOCAL PARALYSIS.

The signs of paralysis of peripheral nerves are as follows:—

CRANIAL NERVES.—*Third Nerve*.—Divergent squint, from paralysis of all the muscles of the eye excepting the superior oblique and external rectus; ptosis, from paralysis of the levator palpebrarum; and dilatation of the pupil, from paralysis of the sphincter of the iris. Diplopia is crossed.

Fourth Nerve.—By paralysis of the superior oblique muscle of the eye, the pupil is slightly raised above the lower lid, and there is no power of rotating the eye. *Test*.—If the patient be directed to look at an object below the horizontal median plane, he sees double, and the image formed by the affected eye is displaced downwards and inclined to one side.

Fifth Nerve.—Ophthalmic Division.—Loss of sensation over one-half of the forehead, the upper eyelid, conjunctiva, and a part of the mucous membrane and integument of the nose. The corneal reflex is lost, and destructive ulceration occasionally follows. *Superior maxillary division.*—Anæsthesia of the lower eyelid, cheek, side of the nose, and half of the upper lips, gums, and palate. *Inferior maxillary division.*—Anæsthesia of the lateral part of the head, face, and ear, half of the lower lip and gums, and half of the tongue, with paralysis of the muscles of mastication. When the lesion is at or near the Gasserian ganglion, the sense of taste is often lost on the same side; and herpes on the face is not uncommon.

Sixth Nerve.—Convergent squint from paralysis of the external rectus muscle of the eye. Diplopia is homonymous.

Seventh Nerve.—Peripheral paralysis of the facial nerve may arise from disease affecting the nerve (1) within the cranium; (2) in its course through the temporal bone; or (3) at its periphery. The first form, which is rare, may be caused by disease of the medulla, or by the pressure of some tumour. The second form is more common and is generally due to disease of the temporal bone following inflammation of the middle ear. The third form is most common, and usually arises from exposure to cold.

SYMPTOMS.—In the ordinary form (affecting the nerve outside the stylo-mastoid foramen), the onset is abrupt. The facial muscles of the paralysed side are motionless and flaccid; the lines of expression are lost; the eyelid cannot be closed, and remains open; the tears overflow the lower eyelid; the cheek is puffed out on forcible expiration; the mouth is drawn to the opposite side, and food lodges between the teeth and the cheek on the affected side. The paralysis becomes at once apparent on the patient attempting to frown, purse the lips, or show the

teeth. The affected muscles waste, and exhibit the reaction of degeneration. There is no anæsthesia.

In the *second form*—over and above the symptoms already mentioned—the nerve to the stapedius muscle, the chorda tympani and the large petrosal branches of the facial nerve are also paralysed. Thus we find disturbance of hearing, flattening of the arch of the palate on one side, with deviation of the uvula to the sound side; and also some impairment of taste, owing to the suppression of the salivary secretion on one side, or to some obscure direct influence of the chorda tympani on the tongue itself.

In the *first form* we may further meet with head symptoms and signs of implication of other cranial nerves.

The supra-nuclear facial paralysis which accompanies hemiplegia differs from the above as follows:—(1) The orbicularis palpebrarum is not markedly involved, so that the eye can be closed as usual; (2) the tongue is also paralysed as a rule, so that it deviates when protruded; (3) the tone of the muscles, and their reflex and electric irritability, are normal; and (4) the paralysis is for voluntary movements only, automatic movements as in smiling being unaffected. The course and duration vary greatly according to the cause and degree of severity. In ordinary cases, recovery is more often complete, after a duration of two or three weeks, or as many months; but it is sometimes incomplete, or paralysis may be permanent.

TREATMENT.—When the paralysis is due to disease of the brain or of the temporal bone, treatment must be directed to these conditions. In the third form massage and electricity are of most service. If the muscles respond to faradism that form of current is the best; but if otherwise, slowly interrupted galvanism should be employed.

Affections of the *ninth* (glosso-pharyngeal) nerve are so rare that no special description need be given here. Paralysis of the *tenth* (vagus) nerve is most common after its

junction with the accessory nerve. The main symptoms are then paralysis of the soft palate, anæsthesia and paralysis of the larynx on the same side. Vomiting, tachycardia, and irregular breathing may also be met with. Laryngeal paralyses have already been dealt with.

SPINAL NERVES.—It is impossible to describe here in detail the various paralyses of spinal nerves. It must be remembered that each nerve conducts both motor and sensory impulses, and hence paralysis is always associated with anæsthesia in the parts supplied by the nerve below the seat of the lesion, and trophic troubles are not unlikely to arise within the area of its distribution. Injury, pressure and direct implication of the nerves in disease are common causes of local peripheral paralysis. It may perhaps be well to mention that paralysis of the *musculo-spiral nerve*, which is of very frequent occurrence from sleeping on the arm over the back of a chair, closely resembles the paralysis which results from lead-poisoning. The history of the case, the association with the paralysis of more or less anæsthesia, and the inclusion of the supinator longus (which almost always escapes paralysis from lead-poisoning) will, however, suffice to distinguish it.

LOCAL NERVOUS SPASMODIC AFFECTIONS.

“**WRITER’S CRAMP**,” or “**SCRIVENER’S PALSY**,” as the terms imply, is especially common with those who write inordinately, and is characterised by intermittent pain, weakness, and spasm of the muscles of the hand and forearm. Analogous conditions occur in violinists, tailors, and others whose occupations involve persistent fatigue of special muscle-groups. Its pathology is obscure.

Symptoms.—Its earliest indication is a painful sense of fatigue and weakness, which comes on shortly after commencing to write. Sooner or later this is accompanied with

clonic or tonic involuntary spasms of the muscles employed, so that the hand-writing is unsteady and scrawly. At first it can be more or less controlled by voluntary effort, but it becomes worse and worse till at last the use of the pen is impossible. Patients then often learn to write with the other hand, but only to find that, after a while, this becomes affected in a similar manner. In the later stages, a neuralgic condition of the whole limb may be set up, which continues after the effort to write has ceased. There is no real paralysis, no wasting, and (apart from the pain) no disturbance of sensation; moreover, for all other acts than that which has induced the disorder the muscles behave properly. The prognosis is unfavourable.

“WRY-NECK,” OR TORTICOLLIS.—Two varieties are met with—congenital, and spasmodic. The former is present from birth, and is characterised by a persistent contraction of one sterno-mastoid (generally the right), associated with facial asymmetry. It may be relieved by tenotomy.

Spasmodic torticollis of the ordinary type is characterised by tonic or clonic spasm of certain muscles of the neck—generally the sterno-mastoid (drawing the head down to one shoulder, with rotation to the opposite side), and the cranio-clavicular part of the trapezius (drawing the head down to the same side), which are both supplied by the spinal accessory nerve. The shoulder on the affected side is very commonly shrugged up. It is a disease of middle-life, especially affecting highly nervous subjects.

It comes on for the most part insidiously, beginning in the sterno-mastoid, and gradually involving other muscles. The trouble is increased by fatigue or excitement, and is relieved by rest. The spasm may be accompanied with considerable aching pain in the side of the head and neck and down the arm.

The treatment is very unsatisfactory. Rest, good feeding, and tonics are chiefly indicated. In aggravated

cases of spasmodic torticollis, excision of a portion of the spinal accessory, combined with section of the posterior primary divisions of the upper cervical nerves, has been practised with success.

SUB-SECTION V.

FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

EPILEPSY.

Epilepsy is essentially a cerebral disorder, and is not necessarily connected with coarse structural change. It consists of recurrent attacks, characterised by temporary loss of consciousness of sudden onset, of short duration, and often associated with convulsion. It commonly makes its first appearance some time before twenty years of age, and less frequently as age advances. There is often a distinct family history of epilepsy or of some other nervous disease, insanity, or intemperance, or again there may be a previous personal history of infantile convulsion, shock, over-strain, or masturbation; but obvious predisposing causes are often wanting. There is no morbid anatomy of epilepsy; all that can be said is that the attacks probably represent an explosive discharge of nervous energy in some part of the cerebral cortex, as the effect of morbid function or nutrition.

SYMPTOMS.—It is customary to recognise two forms of true epilepsy, though the distinction is somewhat artificial, and intermediate cases are of frequent occurrence:—(1) *Minor epilepsy*, or *petit mal*; and (2) *Major epilepsy*, or *haut mal*. A short description will also be given of epileptic attacks which are secondary to a definite irritative lesion of the brain, known as *Jacksonian epilepsy*.

1. MINOR EPILEPSY.—The attack comes on *suddenly*,

without warning, and is of only momentary duration. It is characterised by loss of consciousness, slight pallor, a vacant expression, and dilatation of the pupils. Any voluntary acts in which the patient may be engaged are abruptly stopped, while involuntary acts, such as walking, continue. He does not fall, and recovers himself after a few seconds, often oblivious that anything unusual has occurred. Occasionally after such an attack certain curious automatic acts may be observed, or hysteroid convulsions may occur. The former are important from a medico-legal point of view, as in this condition criminal acts may be perpetrated, of which the patient has no knowledge, and over which he has no control. Thus articles may be stolen or hidden, or assaults committed, or the patient may indecently expose himself, and so on.

2. MAJOR EPILEPSY.—The attack is often preceded by premonitory phenomena, known as the *epileptic aura*. The aura varies widely in character in different cases, but is remarkably constant for the same person. It usually takes one of the following forms:—A movement or sensation of numbness or tingling in the extremity of a limb, epigastric discomfort, a choking sensation in the throat; giddiness, or some disturbance of sight or vision.

The *actual attack* or “*fit*” may be conveniently described in three stages. The *first stage* is often ushered in by a shrill cry, which is followed by absolute and instantaneous loss of consciousness, in which the patient falls more or less violently. *Tonic* muscular contractions of certain parts now supervene, by which the hands are clenched, the limbs twisted, the head and eyes rotated to one side, and the face hideously distorted; the breath is held; the face becomes pale and then livid; and the pupils are markedly dilated. This stage usually lasts from thirty to forty seconds.

In the *second stage* loss of consciousness continues.

Clonic spasms replace the tonic spasm, causing the features to work, the eyes to roll, the jaws to champ, the tongue to be frequently bitten, the limbs and trunk to be more or less violently convulsed, and the pupils to oscillate. As air begins to re-enter the chest lividity becomes less marked; the breathing is noisy and irregular; foam forms at the mouth; profuse perspirations break out; the pulse is full, and the heart beats violently; and both urine and fæces may be involuntarily discharged. This stage lasts from one to three or four minutes, and passes off slowly.

The *third stage* is marked by a gradual return of consciousness. The muscular spasms cease; while the patient opens his eyes, perhaps tries to get up, and looks bewildered. He is very exhausted and mentally confused, and frequently falls into a deep sleep which may continue for some hours. After the attack a large quantity of pale limpid urine may be passed, and sometimes temporary paralysis of the limbs is observed.

The fit may not be repeated; but more often it recurs periodically at intervals of a week, a month, or a year. Attacks may occur by night as well as by day. During the intervals the patient may feel quite well, but the general health is defective as a rule. The memory may fail, the mind become dull, or acute mania or dementia may supervene. Occasionally a large number of attacks occur in quick succession for several hours (*status epilepticus*). The temperature rises, and the patient is reduced to a state of extreme exhaustion, which may be fatal.

The main points on which a *diagnosis* should be based are:—infrequency of first occurrence late in life, possible occurrence in sleep, sudden onset, insensibility of the cornea, dilatation of the pupil, frequency with which the tongue is bitten, frequent injury from falling, involuntary passage of urine or fæces, absence of emotional display, short duration, and subsequent sleep.

Prognosis.—A single attack is rarely fatal, but the disease, if left to itself, tends to persistent recurrence gradually unfitting the sufferer for the duties of life; and we can only speak of cure or amendment in proportion as the attacks are arrested or rendered less frequent by treatment. The cases most favourable for treatment are, *cæteris paribus*, those in which it is begun early, and thoroughly carried out, especially between the ages of 3 and 7, and of 16 and 21 years; when the attacks only occur at night; and where the intervals between them are long. Minor epilepsy, instead of being more amenable to treatment, is far less so.

JACKSONIAN EPILEPSY depends upon irritative lesions of the brain-cortex, commonly in connection with local injury, tumour, old thrombosis, general paralysis, or meningeal hæmorrhage. It is characterised by convulsion limited to a particular group of muscles in the hand, foot, or face, or at least beginning in a particular group; and when extending beyond this, it does so in a definite order. The attack is usually preceded by a feeling of numbness or formication in the affected part. There is no loss of consciousness, the tongue is not bitten, and partial and temporary paralysis of the affected muscles frequently follows an attack. In course of time the convulsion is apt to become generalised; and when this is the case, consciousness may be lost in the *later* stages of the attack—not suddenly at the onset, as in ordinary epilepsy. When the face is the seat of convulsion, it may be associated with temporary aphasia. Analogous local sensory disturbances may occur as the result of irritative lesions of a limited area of the sensory brain-cortex.

Treatment.—The life of an epileptic should be regular, and free from excitement and nervous over-strain of any kind. Plenty of fresh air and exercise are desirable—in short, a mode of life which makes for good general health.

The diet should be simple, and contain only a small proportion of meat. Alcohol and tobacco should be forbidden. The bowels require frequent attention. Enlarged tonsils and post-nasal adenoid growths should be removed. Of drugs the bromides of potassium, sodium, or ammonium are pre-eminently the most useful, and should be given in full doses from one to three times a day; to be continued for at least two years after the last attack, and then gradually suspended (Gowers). In order to prevent the acniform eruption caused by bromide, combination with arsenic is desirable (liq. arsenicalis ℥ij.-iv., three times a day). If the attacks are not arrested by bromides, the addition of tincture of belladonna (℥v.) should be made to each dose of the bromide mixture; or a trial may be made of sodium biborate (gr. v.-x. thrice daily), or the salts of zinc. The latter have more influence on minor attacks (Gowers). Opium in any form should be avoided. In Jacksonian epilepsy with a syphilitic history, a course of mercury and potassium iodide is desirable; and in cases where a superficial cortical lesion can be localised, the propriety of trephining should be considered.

During a fit, but little can be done beyond loosening the clothing, and gentle restraint to prevent injury.

INFANTILE CONVULSIONS.

The undeveloped state of the cerebral cortical centres in infants renders them peculiarly liable to convulsion from disturbance of various kinds, especially when their nervous instability is emphasised by inherited or acquired feebleness of health, or by rickets. The commonest exciting causes are sudden accession of a specific fever, exhaustion from profuse diarrhœa, mechanical congestion of the brain as in paroxysms of whooping-cough, and reflex irritation from indigestion, worms, or dentition.

The *symptoms* are not quite the same as in epilepsy. The attacks are usually less severe, but of longer duration, and the loss of consciousness is less complete. Often they are limited to slight twitching of the eyelids, mouth, face, or one limb; accompanied by a blank stare, or deviation of the eyes; with the fingers flexed, and the thumbs turned in across the palms (known as "*inward fits*"). Or the body may become stiff, with temporary arrest of breathing and consequent lividity, followed by general clonic convulsion. If unilateral, some organic cause should be suspected. After a variable time the spasms cease, and the child falls into a deep sleep. Several attacks may occur in rapid succession, attended with coma, which may prove fatal. It should be borne in mind that every convulsive attack renders the child more prone to another; that convulsions in infancy create a tendency to epilepsy in later life; that they interfere with the nervous development of the child, producing a liability to various functional nervous disorders; and lastly that, when severe, or oft-repeated, nervous development may be altogether arrested, and lead to idiocy.

We should not too hastily ascribe such convulsions to gastro-intestinal irritation, but should consider the possibility of some acute febrile disorder, search for evidence of rickets, and look out for signs of meningitis from tubercle or ear disease.

In simple infantile convulsion, the main indication is to search for possible causes, with a view to their removal. With regard to immediate treatment, a warm bath may be given; and, if the child's bowels are not already well open, a dose of castor oil, or a grain of calomel is desirable. When the convulsion is severe or unduly persistent, it is well to give a rectal injection of chloral and bromide of ammonium (about 3-5 grs. of each for a child of a year old), and repeat it as required.

HYSTERIA.

Hysteria is a peculiar form of nervous disturbance and perversion, which cannot be referred to any substantive anatomical change in the nervous system. It is intimately associated with general nervous instability, and is characterised by a tendency to dissociation of personality, of such a kind that the lower types of nervous function are not normally gathered up, ordered, and controlled by the higher psychical centres.

Hysteria usually first appears at some time between puberty and twenty-five years of age, and chiefly (though not exclusively) in females. Of the causes of hysteria we know very little. The general nervous defect underlying hysteria is undoubtedly inherited; but shock, emotional excitement, sexual disturbance, and the absence of definite occupation, one or all, may have an important influence in the development of its clinical manifestations. There is no necessary connection between hysteria and sexual disturbance, but when we bear in mind the large share which such functions assume in the physiological constitution of the female, it is not surprising that any profound impression upon the one should be associated with disturbance of the other.

The *symptoms* of hysteria may be conveniently arranged in groups, which may occur separately, or in every possible combination.

1. The "*hysterical fit*" is a convulsive paroxysm, of *gradual* onset, beginning with sobbing, laughing, or other emotional displays, and often associated with a feeling of a ball in the throat (*globus*), until perhaps the patient sinks to the ground, apparently insensible. A little later, clonic spasms, varying from a general tremor to more or less violent irregular convulsive movements, may be observed. This continues for a while—with or without intermissions

—when recovery takes place, often accompanied with more sobbing or other emotional display, flatulent eructations, and a feeling of exhaustion. The insensibility is very rarely complete, the movements are of an irregular purposive type, the eyelids are closed and tremulous, the pupils respond to light, the corneal reflex is never lost, the patient does not bite her tongue, never injures herself, there is no foaming at the mouth, no involuntary discharge of urine or fæces, the fit is not followed by coma, and it never comes on during sleep.

2. *Mental Disturbances.*—The controlling power of the will is always much weakened, and thus the emotions, removed from proper restraint, are readily excited by trivial causes. In other cases there is absolute moral perversion, together with extraordinary whims and caprices; also a morbid self-consciousness, and a great anxiety to create interest and sympathy at any cost, which often leads to most outrageous acts of deception. Not infrequently hysterical patients take to drinking in excess.

3. *Sensory Disturbances.*—These consist of (a) *neuralgic pains* with tenderness on pressure in various parts, which are often limited to a single part, *e.g.* the scalp (*clavus hystericus*), the breast (*mastodynia*), the spine, the ovaries, or the joints; (b) various *perverted and morbid sensations*, as noises in the ears, flashes before the eyes, tingling in the limbs, or a peculiar sense of choking or constriction, as if from a ball in the throat (*globus hystericus*); (c) *exaggerated sensibility (hyperæsthesia)*—thus, one or more of the special senses may be excessively acute, or there may be intolerance of light and sound, or the skin may be so sensitive in parts that it cannot be touched without causing pain; or (d) *anæsthesia* of the skin, which is generally limited in extent and does not correspond with the distribution of any special nerve, occurring in well-defined areas, and often affecting a segment of a limb, such as a hand

or foot. In other cases the anæsthesia may affect the whole of one side of the body, and be associated with restriction of the field of vision and colour-blindness in the eye of the affected side. (See p. 560.)

4. *Motor Disturbance. Spasm.*—In addition to the convulsive attacks already described, we occasionally meet with more or less persistent flexion of the arm, spastic extension of one or both legs or feet, or trismus. Tremor and rhythmical movements of the limbs are also sometimes observed. Hysterical spasticity is never attended with an extensor plantar reflex. *Paralysis.*—This is of very common occurrence, and may appear as paraplegia or hemiplegia, or be limited to one limb, or to a particular region—as in aphonia from paralysis of the laryngeal muscles. Paralysis frequently follows an hysterical fit, or emotional outburst of some kind. In paraplegia, the legs may be moved when lying down, though the patient is unable to walk; the nutrition of the muscles is good, and electric reactions are normal. Anæsthesia, if present, is generally irregularly distributed. Exaggerated knee-jerk with absent plantar reflex is very suggestive; and the latter is never extensor. The sphincters are not involved. In hemiplegia of hysterical origin, the face is almost never affected; the leg on the affected side is dragged helplessly along the ground, and is not swung round; the knee-jerks are equal on both sides; and hemi-anæsthesia is often well marked. (See p. 560.)

5. *Visceral Disturbance.*—Under this heading are included palpitations, hurried breathing, barking cough, depraved appetite, digestive derangements, flatulence, persistent hiccough, and constipation, any one of which may be present. Retention of urine is of common occurrence, but incontinence never happens.

TREATMENT.—Firm and considerate behaviour, assisted in an emergency by a douche of cold water, will generally

suffice to rouse the patient from the fit. In bad cases a subcutaneous injection of apomorphia may be given, in a dose not exceeding gr. $\frac{1}{10}$.

During the intervals between the fits the great aim should be to get the patient's confidence and respect, the moral effect of which will often alone determine a cure, and will always enhance the favourable influence of any other treatment which may be employed. In severe cases, it is of the utmost importance to remove the patient from her usual surroundings, and to transfer her from the care of her friends to that of a professional nurse. For all kinds of undue excitability, bromide of potassium is very useful, and may be combined with Indian hemp or valerian; for paralysis—strychnia, phosphorus, and faradisation; for anæsthesia—faradism with the wire brush. The importance of attention to the general health and mode of life cannot be over-estimated. As a general rule, alcoholic stimulants do more harm than good with hysterical subjects.

PARALYSIS AGITANS.

Paralysis agitans, or Parkinson's disease, is chiefly characterised by trembling. Nothing is definitely known respecting its causation or morbid anatomy. It is rare under forty years of age; and it affects men twice as often as women.

SYMPTOMS.—The onset is generally very gradual, and is usually first manifested by *trembling*, which begins in one hand. It is at first quite slight and intermittent; but later on, it causes a characteristic rubbing or rolling of the thumb upon the fore-finger. This is often associated with alternate pronation and supination, or alternate flexion and extension of the wrist. The movements con-

tinue during repose, but cease during sleep; they are increased by excitement, but may be temporarily controlled by an effort of the will. After a variable and often protracted interval the tremor extends to the leg of the same side, and later to the arm and leg of the opposite side. Tremor sometimes extends to the muscles of the neck and face, but the trunk is very rarely involved. As a rule, *weakness* of the affected muscles is not observed till the later stages, though it may set in earlier, and when once it has begun it slowly gets worse. Meanwhile, the affected muscles display more or less *rigidity*, which brings about a characteristic posture, gait, and expression. Thus, the neck and body are stiffened and inclined forwards, the forearms are flexed and the hands carried in front of the body, and the legs also are bent at the knees. When moving, therefore, the patient has a tendency to run, as if to save himself from falling forwards, taking short shuffling steps, and scarcely raising his feet from the ground (*festination*). The features become fixed and expressionless (Parkinson's mask). All movements are performed slowly and with effort. Speech becomes high-pitched, monotonous, and finally reduced almost to a whisper. The reflexes are usually normal, and the electrical behaviour of the muscles is unaltered. The course is very chronic, extending over many years, and death more often occurs from intercurrent disease.

In typical cases the diagnosis is fairly easy. The character of the tremor, and the absence of nystagmus, optic atrophy, sphincter defects, and extensor plantar reflex serve to distinguish it from disseminated sclerosis. Neurasthenia sometimes causes difficulty, especially in those rare cases of paralysis agitans in which there is no tremor. Senile tremor chiefly affects the head and neck, often occurring only on attempted movements, and rigidity is a less prominent feature.

The *treatment* is very unsatisfactory ; but arsenic, cannabis indica, and opium have been recommended.

CHOREA.

Chorea is a functional disorder of the nervous system, characterised by disorderly movements of a peculiar kind. It is most frequently met with between six and twenty years of age, more often in girls than in boys. It sometimes affects pregnant females, and isolated cases occur in advanced life—with or without organic nervous disease. Nothing is definitely known as to the pathology of chorea ; but there is no doubt that, whatever its proximate cause, a condition of general nervous instability underlies it. The relation to rheumatism is very striking. So close and frequent is the association between these two conditions, that chorea is regarded by some as a definite rheumatic manifestation (cerebral rheumatism). Chorea may develop in the course of acute rheumatism, many choreic patients have previously suffered or subsequently suffer from that disease, and inflammatory cardiac troubles are frequent in both. Antecedent scarlet fever is sometimes noted, and the occasional association with pregnancy has already been referred to. A certain proportion of cases may be proximately traced to shock, excitement, or mental over-strain, but more often no adequate exciting cause can be discovered. No constant anatomical changes in the central nervous system have been identified.

SYMPTOMS.—In the mildest form, most common before puberty, there is at first general fidgetiness, with nervousness, disturbed sleep, and some emotional excitability. Then the child is observed to be awkward in its movements, apt to jerk the limbs, make grimaces, or drop things from the hand. Before long, the characteristic involuntary irregular movements occur, either limited to the face,

hand, or to the limbs of one side (*hemi-chorea*), or affecting all of these parts simultaneously.

In severe cases, more common in children after puberty, the movements are much more violent. The features work incessantly, the eyes roll, the lips are in turn separated and pursed up, the tongue is now and again thrust from the mouth, the head is jerked from side to side, the limbs (especially the arms) are variously and irregularly flexed, extended, or adducted, so that walking or even standing may be impossible; and the trunk may be tossed about so violently, that the child can scarcely be kept in bed. Food can only be taken with great difficulty. The diaphragm shares in the general commotion, and the breathing is irregular and unequal. The heart's action is very rapid, and sometimes irregular. Speech is difficult or altogether impossible. The movements are intensified by any voluntary act, by excitement, or when watched; but they cease during sleep. The affected muscles are always more or less weakened. The knee-jerks are sometimes of a tonic type, *i.e.* the resulting muscular contraction is unduly prolonged. The expression of the face is blank and fatuous; and in cases of long duration, there may be actual mental defect. With regard to the general health, the appetite is bad or capricious, the bowels are confined, and nutrition is impaired. Marked anæmia is also very common. There is no fever as a rule, in uncomplicated cases.

In many cases, the movements may be temporarily inhibited by voluntary action; but under these circumstances, it is not uncommon to find evidence of inco-ordination, in an inability properly to control the muscles concerned in the act, as in writing, for instance—and this in spite of complete cessation of all disorderly movements. Voluntary movement may further be interfered with by muscular weakness, especially in cases of *hemi-chorea*, in which there is generally less spasm. It

never amounts to actual paralysis. It may develop gradually in the course of the attack, or in some cases before choreic movements have begun, or persist for a while after they have ceased. In other cases, fortunately not common, maniacal symptoms may supervene, in association with profound prostration, fever, and the condition known as the "typhoid state."

Cardiac murmurs are very frequently observed in chorea, and present one or more of three varieties :—basal murmurs of hæmic origin, murmurs due to mitral regurgitation from relaxation of the ventricular wall, and murmurs due to endocarditis.

The average duration of chorea is from four to six weeks ; but relapse and recurrence are very common. The majority of cases recover ultimately, but the maniacal form is not infrequently fatal. Some mental dullness may continue for a time after chorea.

TREATMENT.—Rest in bed and strict avoidance of excitement are absolutely essential in all but the mildest cases. Any discoverable source of irritation should be removed if possible. A brisk purgative is useful at the outset.

The drugs employed may be divided into two classes, viz. : *sedatives*, as chloral and bromide of potassium ; and *nervine tonics*, as arsenic, zinc, iron, and silver. In acute severe cases it is well to bring the patient thoroughly under the influence of chloral for some few days, giving food in the short intervals between sleep. It should be remembered that acute chorea is a depressing and exhaustive disease, and a liberal supply of nutritious food is not only desirable but essential ; and if the requisite amount cannot be given by the mouth, it must be supplemented by nutrient enemata. In severe cases, with a tendency to lapse into the "typhoid state," alcohol should be freely administered. As improvement takes place, sedatives must give place to tonics ; and in ordinary cases such

remedies may be used from the first. Arsenic is the most useful, and may be given alone, or combined with steel wine, or the ammonio-citrate of iron. A very good plan of using arsenic is that recommended by Dr. Murray, namely, to give large doses—say ℥xv. of iq. arsenicalis, three times a day—well diluted, from the outset. It can generally be borne for about a week, when the dose should be reduced. Phenazone, in doses of gr. v. or more, thrice daily, and gradually increased, has also been strongly advocated. In chronic cases cod-liver oil, general tonics, good food, and change of air are of most service. For persistent weakness of the limbs, after the movements have ceased, strychnia is of considerable value, and friction with faradisation of the limb should be employed and the patient encouraged to use it.

TETANY.

Tetany is not common even in its mildest forms, and severe cases are decidedly rare. It is characterised essentially by generalised over-excitability of the neuro-muscular apparatus, manifesting itself by paroxysmal attacks of tonic spasm. It is met with most frequently in children, with whom it is generally of a mild type; but it also occurs in adults, especially in connection with gastro-intestinal disturbance, pregnancy, infectious fevers, and certain toxic conditions. An epidemic form is also recorded. Its pathology is obscure, but recent research strongly suggests that it is associated with a lack of calcium salts in the body, which in turn depends upon perverted parathyroid activity. It would appear that in some way the parathyroids control the calcium exchange in the body.

SYMPTOMS.—These vary somewhat in different cases, but the two essential features are (*a*) tonic muscular spasm, and (*b*) evidence of increased neuro-muscular irritability.

The tonic muscular spasm chiefly affects the extremities—the upper more often than the lower, and when both upper and lower extremities are involved, the spasm is more marked in the former. The arms are drawn across the chest, the forearms and wrists are strongly flexed, and the hand assumes the “obstetrical” position, with the thumbs turned in across the palms. The feet are extended and turned in, and the toes are flexed. The contractions are often attended with pain, which may be severe. In some cases, the spasm involves the face, or muscles of the trunk. An example of the latter was seen by the writer, attended with marked disturbance of breathing. Increased neuromuscular irritability is shown by increased activity of tendon-jerks; increased electric excitability both of muscles and nerves; and by the fact that, in the intervals between the attacks, spasm may be induced by mere mechanical stimulation of a motor nerve trunk, as by tapping with the finger (Chvostek’s sign). Sensory disturbances are common, such as tingling or creepy sensations in the affected parts. There is no fever as a rule, but in severe cases, the temperature may rise one or two degrees during the attack.

Attacks may be induced by over-exertion, exhaustion, gastro-intestinal disorder, or emotional excitement. Premonitory tingling or numbness often occurs. The spasms persist from a few minutes to an hour or more, and may recur at variable intervals for weeks or months, then passing off. In young children the turning in of the thumbs and flexion of the fingers often persist during the intervals between the attacks of more general spasm. The prognosis for the most part is favourable.

The *treatment* consists in the removal as far as possible of any source of irritation or excitement, simple diet, and the internal administration of calcium salts such as the hypophosphites, or glycerophosphates. Parathyroid pre-

parations appear to be well worthy of trial, but the author has no experience of their value.

SUN-STROKE.

Sun-stroke, or insolation, is the term applied to nervous disturbance arising from prolonged exposure to a high temperature. The temperature of the body is practically constant, and this uniformity is due to equilibrium between heat-formation and heat-loss. Under the influence of the sun's rays the thermo-regulating nervous mechanism is upset, the body becomes overheated, and thus accounts for the symptoms which characterise sun-stroke. This effect is enhanced by heavy or tight clothing, severe exertion, or deficiency of drinking-water.

SYMPTOMS.—Sometimes the onset is sudden, the patient falling down insensible and collapsed, with gasping respiration, extreme feebleness of pulse, and *marked elevation of the temperature*. This form is generally rapidly fatal.

More often the attack is less abrupt, and insensibility (if it occurs) is preceded for a variable time by a feeling of profound exhaustion, headache, restlessness, nausea, and vomiting; or the latter symptoms may occur alone, without insensibility. The temperature is high, and accompanied by febrile symptoms of the usual kind. In severe cases the pulse becomes increasingly quick and feeble, and the breathing hurried and irregular, until the patient lapses into profound and fatal coma. In fatal cases, death generally takes place within forty-eight hours, and may be preceded by delirium or mania, or more rarely by convulsions. In less severe cases recovery more often occurs, but troublesome nervous symptoms may be left behind.

Physical exertion in the presence of high temperature, as with stokers in large steam-ships, is liable to be followed

by extreme prostration and collapse; but it is distinguished from sun-stroke by the coolness of the body-surface, a sub-normal temperature, and the absence of coma or delirium. If death occurs, it is through heart failure, but recovery is the rule.

TREATMENT.—The early employment of cold water is of the first importance, either in the form of a cold bath, a douche to the head, or a wet sheet-pack. The bowels should be freely opened by enemata, and good nourishment is afterwards necessary.

NEURASTHENIA.

It is easy to criticise the use of this term from a strictly pathological standpoint, but it is clinically necessary to adopt some such title to indicate the condition of those who, either as the result of inherited weakness or acquired exhaustion are unequal to the strain and wear-and-tear of life, on a plane of average activity. Among the causes which give rise to neurasthenia, worry, anxiety, and grief undoubtedly occupy the front place. In other cases it may be due to combination of physical and mental shock, as the result, for instance, of a railway accident (traumatic neurasthenia); or to abuse of drugs, especially morphia and cocaine; or to exhausting and depressing disease, such as cancer, chronic renal disease, or influenza. Neurasthenia is obviously more likely to occur in those who have, by inheritance, a weak and unstable nervous constitution; but exactly similar results may follow excessive strain in a person of normal nervous constitution. In the former class neurasthenic disturbance is also likely to occur earlier in life than in the latter. Men and women are equally liable.

The *symptoms* of neurasthenia vary much in detail in different cases, but the following are the most common. In the first place, there is a general incapability for both

physical and mental exertion. All pleasure in work is lost, fatigue soon follows, thoughts wander, and concentration of thought and interest become difficult or impossible. Patients lose self-confidence; they shrink from responsibilities for fear of failure; they become morbid, self-centred, and depressed, and are prone to emotional manifestations on slight provocation. One of the commonest symptoms is occipital headache with pains extending to the neck, with a sense of vertical pressure, or as if the head would burst. Giddiness is a frequent complaint, often accompanied with sinking feelings. Sleep is restless and disturbed. It is common for a patient to fall asleep soon after getting to bed, to awake in the course of an hour or two, then to lie fretting and tossing about for a long while, then drifting into broken slumber, and finally to awake feeling tired and quite unrefreshed, with no appetite for breakfast. The appetite may be better for later meals, and it is usual for neurasthenics to improve as the day goes on, and to be at their best in the evening. Digestive disorders with flatulence, motor insufficiency, and constipation, sometimes accompanied also with displacements of abdominal viscera, are of very frequent occurrence, reacting unfavourably upon the nervous condition, and tending to establish a vicious circle which is very difficult to deal with. Among other common symptoms are tenderness of the spine with radiating pains in various directions, numbness and tingling of the extremities, cold hands and feet, flushings and heats, tremor, palpitation, frequent low-tension pulse, nocturnal emissions, and sexual excitability with lack of sexual power. In traumatic neurasthenia there may be a considerable interval between the primary shock and the onset of symptoms. For a full account of the profound psychoses of a hysterical type which may occur in traumatic cases, the reader is referred to standard works on the subject.

With due care the *diagnosis* of neurasthenia should seldom be difficult. The possibility of hitherto undiscovered organic disease, such as cancer, diabetes, tuberculosis, or Bright's disease, should always be kept in mind. In distinguishing neurasthenia from organic nervous disease, careful examination of the eyes and reflexes will yield valuable help. In neurasthenia we never meet with squint, diplopia, nystagmus, optic atrophy, or optic neuritis. While the knee-jerks are often exaggerated, they are never absent, and true ankle-clonus and extensor plantar reflex never occur. The peculiar behaviour of the muscles to faradism will serve to distinguish myasthenia gravis. Distinction from hysteria may be difficult, especially as the two conditions may be combined; but the greater frequency of the latter among women, the extravagant demeanour, the rapid variations, and distinctive hysterical symptoms such as convulsion, paralysis, and objective anæsthesia will generally prevent mistakes.

For this condition there is only one remedy—rest for body and mind, with abundant nourishment; the practical question is how best and most efficiently can these principles be acted upon. Massage and electricity are most useful adjuvants in properly selected cases. The symptomatic treatment necessarily varies according to circumstances.

NEURALGIA.

The term neuralgia is applied to flashes of remittent or intermittent pain in the course of a nerve, unattended by obvious structural change. It may be due to direct irritation of a nerve from exposure to cold, injury, or pressure; or to reflected irritation proceeding from some internal viscus; while in other cases it is a manifestation of some toxic condition, as in gout, alcoholism, or specific fevers. It rarely occurs before puberty, and is most

common in early adult life, especially among women of a nervous temperament. Pathologically, it is very closely related to neuritis; and no doubt, strictly speaking, many of the severer cases are really of an inflammatory nature.

SYMPTOMS.—Wherever seated, neuralgia presents certain characteristic features. The *pain* may be variously described as aching, boring, shooting, burning, etc.; but *it is always more or less paroxysmal*. It is generally severe, and sometimes of horrible intensity. On palpation, one or more *tender spots* are very constantly observed, with an invariable relation for each nerve, and they usually coincide with the exit of the nerve from a bony foramen, or its passage through some dense fascia. The pain is frequently associated with local abolition of tactile sensibility, with twitchings or cramp-like contractions of muscles, with congestion of the affected surface, and with trophic lesions of the skin, especially erythematous and herpetic eruptions. The pain of neuralgia is further characterised by its *tendency to shift from one spot to another*, in the course of the same or some other nerve; and also by being *almost invariably unilateral*. As to the general health, neuralgia *is always attended with debility* from one cause or another, *e.g.* mental anxiety, anæmia, gout, rheumatism, syphilis, and dyspepsia.

Special terms are employed to indicate neuralgia of particular nerves; thus, *tic douloureux* (fifth cranial nerve); *sciatica* (sciatic nerve); *brachialgia* (nerves of the arm); *gastralgia* (nerves of the stomach); and so on. Separate description of each is beyond our purpose. Some forms of visceral neuralgia have already been referred to in the description of cardiac and gastric neuroses.

Neuritis is distinguished by tenderness along the course of the affected nerve, wasting and reaction of degeneration in the muscles supplied by it. It should be remembered that neuralgic pains may be the initial sign of tabes

and certain cases of multiple sclerosis or syringomyelia. Hysterical neuralgia is more, and attended with other hysterical manifestations. The prognosis varies according to the cause, and is more unfavourable in elderly than in young subjects.

TREATMENT.—Whenever possible the cause should be ascertained and an endeavour be made to obviate or remove it. Food should be abundant and nutritious, and should include a good proportion of fatty ingredients. It is scarcely necessary to add that rest and freedom from worry, exhaustion, or excitement are urgently indicated. To relieve the pain we may have recourse to internal or external remedies, or to both combined. Internally, the most important are—quinine, which may sometimes be carefully combined with sodium salicylate arsenic, in combination with iron; tr. of gelsemium (℥ xv.-xx. every four or six hours); phenazone or phenacetin, in doses of gr. v.-xv.; chloral or butyl-chloral. Externally, hot fomentations, counter-irritation, frictions with turpentine or camphor or belladonna or aconite liniment, or veratria ointment, are useful. Should all these fail, hypodermic injections of morphia may be resorted to.

MIGRAINE.

Migraine is a peculiar paroxysmal form of headache, which is otherwise known as megrim, hemicrania, or sick headache. It is markedly hereditary, and affects females more than males. It generally begins about puberty, and rarely after middle life. It is, however, by no means uncommon in younger children. It is peculiarly associated with the nervous temperament, and probably in all cases there is inherited nervous instability. Reflex irritation from carious teeth, uncorrected errors of refraction or astigmatism, post-nasal growths, digestive

derangement, and ovario-uterine troubles are common proximate causes; while worry, fatigue, mental strain, and work under close and insanitary conditions are also frequent factors. The nature of the disease is unknown.

SYMPTOMS.—The attack usually begins in the early morning, on waking, with some visual disturbance such as dimness of sight, transient hemianopia, zig-zag spectra, or flashes of light. In some cases there may be some mental depression or confusion. More rarely, there is some tingling and numbness of the face or arm, or muscular cramps on the same side, or temporary aphasia.

These symptoms are quickly followed by headache, characterised by pain which may be fixed to one spot, such as the temple, or one eye, whence it extends over the whole of one side of the head. In other cases, the headache is frontal. The pain is variously described as dull, sickening, or *throbbing*; and sometimes it is extremely sharp and violent. It is associated with tenderness of the scalp, a sense of heat about the head, and often with some injection of the eye and lachrymation. It is aggravated by noise or bright light. Meanwhile the patient is pale, his extremities are chilly, he both looks and feels exceedingly ill, and the pupils are contracted. The pulse at the wrist is feeble, while on the affected side of the head it is firm and hard. Anorexia is complete. After a while nausea and vomiting supervene, followed by relief of the headache; and the patient falls asleep, from which he awakes practically well. The duration of an attack varies widely. Under ordinary circumstances it is seldom longer than one day, and in abortive attacks it may be much shorter. A duration of two or even three days is not uncommon in severe cases. The disease may recur at intervals for years, but it generally becomes less frequent and severe, and may disappear altogether in later life.

TREATMENT.—Search should be made for sources of

peripheral irritation, with a view to their removal. The nervous system should be spared in every possible way, school hours should be short, examinations proscribed, and the conditions of life should be made as simple and equable as possible. During the attack, relief may be sometimes afforded by strong coffee, citrate of caffeine (gr. v. every three or four hours), or a moderate dose of phenacetin or phenazone. During the intervals, more good may be accomplished by judicious feeding than in any other way—a minimum of animal food, a free supply of vegetables, and plenty of water. Alcohol and tobacco smoking should be avoided. As in all cases there is deficient reserve-energy it is often wise to break the working-day by recumbent rest each morning and afternoon. Medicinally, quinine or arsenic in combination with mineral acids after meals, together with a tumblerful of alkaline water early each morning, is often most beneficial.

RAYNAUD'S DISEASE.

This is a vaso-motor affection, occurring most commonly in women and children of feeble or neurotic habit, and chiefly induced by exposure to cold.

In its mildest form (so-called *local syncope*), the fingers or toes go pale, cold, and dead. The condition passes off in a relatively short time, leaving the part hot, red, and itchy. In the next grade, either as a sequel of the former or occurring independently, one or more of the toes or fingers, or sometimes the ear, becomes livid and swollen, with more or less itching or (in some cases) severe pain (*local asphyxia*). Mild cases constitute the ordinary chilblain. Such attacks occur at variable intervals, especially during the winter months, and then pass off altogether.

In a still more severe grade unhealthy blebs may form,

which leave superficial sores on bursting. Or there may be actual loss of tissue, and the tip of a finger or toe may slough away. Cases have also been recorded, more often in children, characterised by symmetrical patches of gangrene on various parts of the body, and ending fatally in a few days. Association with paroxysmal hæmoglobinuria has occasionally been noted.

The *treatment* is not very satisfactory. The parts should be kept warm by wrapping in cotton wool, and systematic daily exercise or massage of the affected parts is desirable. Galvanism has also been recommended. In the painful forms of local asphyxia, the application of an ointment made of equal parts of iodine ointment and lard sometimes gives great relief. Severe pain may require hypodermic injection of morphia.

SUB-SECTION VI.

SYSTEMIC INTOXICATIONS.

ALCOHOLISM.

The results of alcoholism will be discussed under three heads:—(a) *acute alcoholism*, (b) *chronic alcoholism*, and (c) *delirium tremens*.

Acute alcoholism, or ordinary intoxication, represents the immediate toxic effects of alcohol taken in excess. Its manifestations vary according to quality of the drink, the quantity drunk, and the temperament of the drinker. In general terms, the process represents a progressive suspension of the functions of the nervous centres, beginning with the abolition of voluntary control, and ending (if a sufficient quantity has been taken in the first place) with paralysis of the cardio-vascular and respiratory centres in the medulla, causing fatal coma. Under ordinary

circumstances, the earlier symptoms of intoxication prevent the taking of sufficient alcohol to cause actual death. As a matter of fact, there are no absolute criteria of alcoholic coma, and the diagnosis turns upon the history of the case, and ability to exclude other causes of coma. When in doubt, therefore, as to whether a man is drunk or dying, one should always act as if he were dying.

In *chronic alcoholism*, we have to deal with the persistent effects of alcohol upon the tissues, characterised especially by chronic nervous degeneration, arterial thickening and degeneration, and general retardation of nutritive changes. The most common signs and symptoms of chronic alcoholism are obesity or evidence of malnutrition, flabby and congested features, with a liability to acniform eruptions. The appetite is bad—especially in the morning—the breath is foul, the tongue is coated, together with morning vomiting, flatulence, and other indications of digestive derangement. The implication of the nervous system is manifested by volitional weakness, mental dullness, irritability of temper, emotional excitability, trembling, restlessness, troubled sleep, headache, and vertigo. Later on, structural changes in various tissues and organs may take place, especially in the liver, kidneys, and peripheral nerves. It should also be recollected that the power of resisting morbid influences of every kind is greatly impaired in habitual drinkers, and thus intemperance becomes an important element in the prognosis of all diseases.

Delirium tremens has no connection with acute alcoholism. It never occurs from a single debauch, however unrestrained, and its symptoms are quite unlike those of acute alcoholic poisoning. It is a feature or phase of chronic alcoholism, related to persistent and oft-repeated drinking rather than to the quantity of liquor consumed in a given time. Of its exact nature we have no precise

knowledge ; but in the writer's opinion, the phenomena of delirium tremens are caused by some poisonous products of perverted metabolism resulting from persistent impregnation of the tissues with alcohol. According to this view, delirium would occur, either when the poison is formed faster than it can be eliminated, or when there is any interference with the ordinary course of elimination.

There is often some premonition of the approaching disorder. The patient becomes irritable and restless, he is wakeful, and his sleep is troubled with horrid dreams, his volitional power is impaired, he loses his appetite, and his bowels are confined.

The actual attack is ushered in by extreme prostration, and complete insomnia. The mind is confused and excited, and soon begins to wander. The delirium is of a peculiar kind. It is of a busy garrulous type, the patient is suspicious of all around him, he sees repulsive animals around him, he fancies himself watched by enemies, or threatened by disaster. He is perpetually picking at the bed-clothes, or trying to get out of bed ; and if not watched he may jump through the window, or attempt to injure himself or others. Occasionally he is wildly maniacal, while in other cases he is sullen and morose. The muscles of the limbs and body generally tremble and twitch, especially on the least attempt at voluntary movement. The body is prostrated, the face is pale or congested, the skin is clammy, the temperature is moderately elevated, the tongue is furred, the breath is foetid ; there is loss of appetite, with thirst, and constipation ; and the pulse is soft, and dicrotous. The above symptoms are usually worse towards night.

Most cases terminate favourably about the third or fourth day, when the patient passes into a sound sleep, from which he awakes with a clear mind, but still very

feeble. On the other hand, in fatal cases, prostration increases, and coma or convulsions may supervene, or the patient may die from sudden cardiac failure. A high temperature is a very unfavourable sign.

It should be borne in mind that slight febrile disturbance, especially traumatic fever and pneumonia, is very apt to determine an attack of delirium tremens in habitual drinkers; and in all cases careful examination should be made for surgical injury or pneumonia, which otherwise might be easily overlooked.

TREATMENT.—For acute alcoholism, the leading indication is to empty the stomach by an emetic or by the stomach tube. As far as possible the patient should be roused by cold affusions, or faradism, and by the administration of hot coffee if he can swallow. In chronic alcoholism, alcohol should be absolutely prohibited in any form; liquid nourishing food should be given frequently; and the bowels should be well opened. Though never to be used in routine fashion, yet great benefit is sometimes derived from the employment of medicinal sedatives. In delirium tremens personal supervision by a competent attendant is essential, and mechanical restraint should not be resorted to unless absolutely necessary. The bowels should be well opened, and nourishing liquid food should be liberally supplied. Alcohol should be entirely withheld, unless there is imminent risk of collapse; and under these circumstances the hypodermic use of strychnia is probably of greater value. Few measures are of more service in calming excessive excitement than a tepid wet sheet-pack, which at the same time acts as a means of restraint. If this fails, morphia (gr. $\frac{1}{2}$) may be subcutaneously injected, but it should be used cautiously. In wild excitement a hypodermic injection of hyoscine hydrobromide (gr. $\frac{1}{200}$) is a useful alternative. During convalescence, quinine and other tonics are indicated.

LEAD-POISONING—PLUMBISM.

Though the susceptibility to the influence of lead varies much in different individuals, the symptoms to which it gives rise are tolerably characteristic when once they have developed. The condition is of frequent occurrence in painters, plumbers, glass-cutters, lapidaries, and others whose employment brings them into habitual contact with lead in some form or other. Drinking-water, cider, or other beverages, which have been stored in leaden cisterns, or conveyed through leaden pipes, may become the vehicle of contamination. Lead undoubtedly accumulates in the tissues, retarding and modifying metabolism, and interfering with the formation of blood. The paralytic symptoms are due to peripheral neuritis.

The subjects of lead-poisoning are pale, sallow, and as a rule ill-nourished, with a liability to digestive disturbances. The *gums exhibit a peculiar blue line* in the margins adjoining the teeth, due to the precipitation of sulphide of lead by sulphuretted hydrogen, which is generated by decomposing matters accumulating round the teeth. Attacks of *colic* characterised by severe griping pains in the abdomen, with obstinate constipation, are of frequent occurrence. The colic is probably essentially of nervous origin, and not directly dependent upon constipation; but its precise cause is obscure. Another characteristic feature of plumbism is paralysis of the extensor muscles of the forearm, causing a *dropping of the wrists*. The extensor metacarpi pollicis, and the supinator longus muscles generally escape. The affected muscles rapidly atrophy, and exhibit the reaction of degeneration. Lead paralysis is always bilateral or tends to become so. Sometimes paralysis invades certain muscles of the arm and leg, and (more rarely) there may be more generalised

weakness, without actual paralysis. Sensory disturbance rarely occurs; but, independently of the paralysis, aching pains in the joints and limbs are occasionally present. Optic neuritis, neuro-retinitis, or optic atrophy may be also observed. Among the rarer forms of nervous disturbance, epileptoid convulsion, hallucinations, and progressive mental failure may be mentioned.

The duration of these symptoms varies greatly, and their recurrence is frequent. The paralysis if neglected is very apt to become obstinate and even incurable. Chronic lead-poisoning is a common predisposing cause of gout, and is often associated with arterio-sclerosis and granular kidney.

An acute form of plumbism sometimes occurs, and chiefly among young women. The patient rapidly becomes anæmic, suffers from headache, and then is suddenly attacked by convulsions, becomes comatose, and dies.

TREATMENT.—Those who work with lead should be careful to cleanse their hands and nails after work, and the habitual use of lemonade with sulphuric acid is probably of service, by converting any lead in the alimentary canal into an insoluble sulphate. In the treatment of lead colic, it is usually sufficient to relieve the pain with opiates and hot fomentations, the bowels generally acting without purgatives in the course of a few days. Should an aperient be thought necessary, an enema or a dose of sulphate of magnesia may be given. Castor oil with opium is also a valuable combination. For the paralysis, local electrification is the only effectual remedy. When taken in hand quite early, faradism will suffice; but if no reaction occurs by this method, slowly interrupted galvanism is far more useful. With a view of eliminating lead from the system, iodide of potassium should be freely administered, together with an occasional aperient draught of sulphate of magnesia in the morning.

MERCURIAL TREMORS.

A peculiar tremor sometimes occurs in connection with chronic exposure to the influence of mercury, which supervenes gradually, usually begins in the arms, and is accompanied with numbness or tingling and pain in the joints. Afterwards the tremors become worse, especially under the influence of any excitement, and then extend to all the voluntary muscles of the body except those of the eyeball. All voluntary movements thus become uncertain, jerky, spasmodic, and feeble, until at last the patient is quite helpless. The tremors cease in the recumbent posture, and during sleep, except at a very advanced period of the disease. After a while, cachectic symptoms supervene, which are sometimes accompanied with grave nervous disturbance. There is no necessary association with mercurial stomatitis or salivation, though they are present, or have previously occurred, in a majority of cases.

If the patient be withdrawn from contamination sufficiently early, the progress is generally favourable in the course of a few weeks. Iodide of potassium, with quinine, iron, or other tonics may be given with advantage.

SECTION XI.

DISEASES OF THE SKIN.

GENERAL PATHOLOGY.

THE exposed position of the skin renders it peculiarly liable to mechanical and chemical irritation, and to damage from physical agents, such as heat, light, and electricity. For the same reason it is very liable to inflammatory damage from parasites—both animal and vegetable—gaining access from without. In another group of cases, eruptions may be caused by micro-organisms which attack the skin from within, such as the rashes of the infective fevers.

Again, many affections of the skin are of constitutional origin, associated with functional disturbance of some internal organ; and, in some cases at least, are probably due to the irritating effects of some morbid material peculiar to the disease in question, excreted by the skin. Eruptions due to drugs are also set up in a similar way. Other diseases of the skin may be partly constitutional, and partly local in origin. Boils, for instance, are caused by the local action of micro-organisms, but with a close relation to habits of life, digestive behaviour, and the state of the general health. The question of constitutional or local origin has an important practical significance, in providing the practitioner with proper indications for treatment.

Other disorders of the skin are traceable to nervous

disturbance, and may be either inflammatory or degenerative in their nature.

Developmental abnormalities are responsible for certain cases, such as the varieties of *nævi*, popularly known as moles and “port wine” marks.

The skin may also be the seat of new growths, innocent or malignant, the pathology of which is as obscure as for similar formations in the body generally.

Any satisfactory classification of diseases of the skin should rest upon a pathological basis, taking into account all the foregoing facts; but this is impossible in the present imperfect state of our knowledge.

NOMENCLATURE.

Apart from the names assigned to individual diseases of the skin, special names are given to cutaneous lesions according to their general character. These form the alphabet of dermatology and must be thoroughly grasped. They are included in two principal groups, according as they are primary or secondary. *Primary lesions* include the essential characters of an eruption, in direct relation to their primary causes. *Secondary lesions*, on the other hand, may or may not be present, and depend upon circumstances of a somewhat adventitious or secondary nature.

The chief primary lesions are small circumscribed areas of skin, altered in colour, and sometimes slightly raised, but not otherwise affected, such as the rash of measles, known as *macules*; circumscribed patches of subcutaneous œdema forming *wheals* or *nodes*; solid local elevations of the skin, forming *papules* or *tubercles*—the former term being conventionally restricted to elevations no larger than a pea; rounded elevations of the superficial layer of the skin containing fluid, which are termed *vesicles* when small, and *bullæ* when of larger size; and similar elevations containing pus, known as *pustules*. Superficial

hæmorrhages into the skin are called *vibices*, *petechiæ*, or *ecchymoses*, according to their shape and size; and *squamæ* or *scales* are flakes of dry and partially detached epidermis. Scales are sometimes due to flakes of dried secretion, but these properly belong to the secondary group.

The chief secondary lesions are *secretion scales*, *scratch marks*, *ulcers*, *pigmentations*, and *scars*. These terms require no special explanation. *Excoriations* follow the rupture of a vesicle or pustule, and are similar to an ulcer except that the loss of tissue is confined to the epidermis. *Rhagades* or *fissures* are deep linear cracks in the skin. *Scabs* are due to dried discharges of serum, pus, or blood; and only differ from scales in their greater thickness and closer connection with the skin.

SYMPTOMS OF SKIN DISEASES.

The disturbances of sensation produced by skin diseases are not numerous; the patient may suffer from burning, tingling, hyperæsthesia, anæsthesia, neuralgic pains, and *itching*.

Itching is a very common symptom, and is especially prominent in scabies, dermatitis herpetiformis, eczema, urticaria, lichen planus, prurigo, and pediculosis. It is usually slight or absent in the syphilides, seborrhœa corporis, psoriasis, and lupus. The patient usually suffers most when in bed, and this is especially the case in scabies; the explanation probably being that his mind is then less distracted by other matters. Itching may also be due to toxic materials circulating in the blood, as is the case in jaundice.

The treatment of itching depends upon the discovery of its cause. When the cause cannot be discovered or removed, we have to fall back upon symptomatic treatment by means of sedative applications or the use of analgæsic drugs internally. External applications are best

used in the form of lotions; the most important being dilute solutions of alkalies, liquor carbonis detergens, carbolic acid, or creolin. Cocaine may be used in severe cases, but is somewhat dangerous, as absorption may take place. Sometimes itching may be relieved by free sweating caused by vapour baths, or by an injection of pilocarpin. The most useful anodyne drugs are cannabis indica and opium, but they should never be used in a routine fashion.

ERYTHEMA.

The term erythema (a blush) simply indicates an active hyperæmia of the skin, and is applied to a series of superficial inflammatory affections of that structure in which redness is the most obvious feature. In some of these (*e.g.* erythema simplex) inflammatory redness forms the whole of the affection; in others (such as erythema iris and erythema multiforme) the inflammation goes further than this, and leads to oedema or the formation of vesicles, hæmorrhages, and pigmentation.

ERYTHEMA SIMPLEX is characterised by a hyperæmic area of a vivid scarlet or pink colour, which varies much in size, is not raised, and has ill-defined margins. The chief symptom is a burning rather than an itching sensation in the region affected, and constitutional disturbance is slight or absent. The latter fact serves to distinguish it from erysipelas. Erythema may be followed by slight desquamation.

Simple erythema may occur from a variety of causes. It may result from exposure to the sun (*E. solare*, or *sun-burn*), the fire (*E. ab igne*), cold (*E. pernio*, or *chilblain*), or from the local action of irritants (*E. venenata*), of which the redness which follows a mustard plaster is a familiar example. Causes which act from within are also numerous. Thus it may occur from certain foods such as shell-fish,

digestive derangements, or after the exhibition of drugs such as atropine, or diphtheria antitoxin, or after the administration of an enema.

The treatment primarily depends upon the discovery of the cause and its removal. A general tonic is often advisable, such as strychnine, quinine, or arsenic (if given with care). Iron is often of value if the condition of the alimentary canal admits of its exhibition. The local treatment consists in the protection of the inflamed region, and the application of remedies which relieve itching. Dusting powders are useful, and the lotions mentioned under the head of pruritus are also applicable. Ointments are not so generally useful, but they are sometimes preferred by patients—one of the “cold creams” (*i.e.* ointments containing much water) will serve.

ERYTHEMA INTERTRIGO is caused by the rubbing together of two surfaces of skin, especially when moist from excessive sweating. It may persist as a simple erythema, but very commonly there is excoriation with much serous discharge, and a true eczema may be thus set up. The treatment is simple and efficacious. The region is to be washed with a dilute antiseptic lotion, dried, and dusted with a drying powder. This latter may be placed in a bag of soft muslin or butter-cloth placed between the opposed surfaces.

ERYTHEMA PERNIO is generally an indication of a feeble circulation. Dusky red patches appear on the dorsal surfaces of the fingers or toes, which itch and tingle severely, especially in the presence of warmth. It should be treated by means of exercise (especially dancing and skipping), local massage and friction, and by cardiac tonics and iron. Stimulating liniments, such as lin. camph. co., or lin. terebinth. aceticum, should be rubbed in vigorously night and morning when the skin is unbroken. Ichthyol is also a useful external application.

ERYTHEMA NODOSUM is common in young women, especially in those with bad general health and in those who suffer from rheumatism. The eruption consists of nodes which generally occur on the front of the legs, and are almost always symmetrical. The areas are oval in shape, about one or two inches long, with their long axes parallel to that of the limb, and margins which fade gradually into the surrounding skin. The nodes are at first raised and red in colour, but afterwards become flatter and exhibit the colour-changes of bruises. Fresh crops of nodes appear from time to time, and the eruption usually lasts about a month.

Rest and tonics are especially indicated, and salicylates should be given internally if there is evidence of rheumatism. Hot fomentations are often agreeable.

ERYTHEMA MULTIFORME is characterised by its variability, and by the fact that papules, nodes, vesicles, bullæ, develop on the primary erythema. These lesions are sometimes further complicated by hæmorrhages. The initial lesion is generally a small papule (*E. papulatum*) of a dusky colour, and leaving a brownish stain on pressure in its later stages. They appear upon the backs of the hands and feet, and extensor surfaces of the limbs. The further development varies. It may simply enlarge (*E. tuberculatum*), or it may extend at the circumference, and clear up in the centre (*E. annulare*), or the rings thus formed may intersect (*E. gyratum*), or give rise to wandering red lines (*E. marginatum*).

ERYTHEMA BULLOSUM is a bullous, and *E. PURPURICUM* a purpuric, form of the disease. A special variety of vesicular or bullous erythema called *Erythema Iris* may be separately described.

ERYTHEMA IRIS occurs symmetrically on the backs of the hands and fingers, the wrists, elbows, and insteps; sometimes also on the nape of the neck. A small reddish disc

first appears, on which a central vesicle or bulla develops, which becomes depressed as the disc increases in area. Outside this a raised white ring develops, surrounded by a pink area. This arrangement of concentric rings is typical of the affection. Severe forms may occur with serious constitutional disturbance; and the mucous membranes of the mouth, tongue, or palate may be attacked. There is a great tendency to recurrence, which may take place at more or less regular intervals for years.

The pathology of erythema multiforme is not known. It is probably due to toxic substances, either developing in the body or introduced from without. There are reasons for thinking that in certain cases it is due to infection with some specific micro-organism.

The treatment should be directed to the removal of any known cause. Gastro-intestinal irritation is so frequently present that it is often desirable to give a mercurial or saline purge, and intestinal antiseptics may also be of use.

In cases associated with rheumatism, sodium salicylate or other antirheumatic drugs may be employed with great advantage. Febrile and severe cases require rest in bed and a light diet. The most useful local applications are lead and calamine lotions.

ROSACEA.

ROSACEA OR ACNE ROSACEA commences as a simple and transitory erythema of the central parts of the face, and in its early stages may be looked upon as an exaggeration of the flushing of the face which so often occurs during digestion. In the second stage of the disease the vessels of the affected area become permanently dilated: and sometimes a third stage occurs, which is marked by great thickening of the skin and subcutaneous tissue. The nose is chiefly affected, the end and sides of which may form

large irregular fibroid tumours known as rhinophyma or "grog-blossoms." The sebaceous glands share in the general hypertrophy of the tissues and may become inflamed, while pustules—unlike those found in *acne vulgaris*—develop without being preceded by the formation of comedones.

Rosacea affects females more than males, and rarely occurs before twenty-five years of age. It has a very distinct and important relationship with dyspepsia, and exposure to the weather. In spite of popular tradition, alcohol only ranks as a predisposing cause in a minority of cases. Seborrhœa capitis is not uncommonly associated with rosacea, and some consider that the latter affection is essentially of a seborrhœic nature.

TREATMENT.—In all cases the digestive system must be carefully regulated, and all stimulating and highly seasoned food, together with tea and alcohol, should be avoided. Constipation and menstrual disturbance may demand attention. In the first and second stages ichthyol is the most useful therapeutic agent: it should be applied locally in the form of lotion or ointment and may also be given internally in pills or capsules. In the third stage surgical interference is necessary: the dilated vessels should be destroyed by electrolysis or by puncture with a hot needle, and the pendulous masses may be excised.

URTICARIA.

The characteristic feature of urticaria is the formation of *wheals* or localised patches of œdema of the hypoderm. The earliest appearance is a pink macule of variable size: this becomes raised and of a white colour, but the erythema may persist at the periphery. It is accompanied by intense itching and burning. Each wheal appears suddenly and lasts only a short time, but there is great liability to recurrence. No part of the body surface is exempt.

The wheal as a rule is due to the action of a poison which dilates the vessels and leads to exudation of serum into the lymphatic spaces. The itching is due to irritation of the nerve-endings. This poison may gain access from without: the wheals produced by the sting of nettles, mosquitoes, fleas, jelly-fish, etc., are familiar examples. Or it may be present in the circulating blood, being derived from the products of dyspepsia, or from certain ingesta, such as *shell-fish* and certain medicinal drugs. Urticaria may also occur in gout, and after the use of antitoxins.

Urticaria papulosa, or *Lichen urticatus*, occurs in children, and is a stubborn and chronic affection. A small irritable papule develops on the top of each wheal, which persists after the wheal has subsided.

Urticaria pigmentosa is not a true urticarial affection. It consists of moderately large flat nodules or macules, which become urticarial when slightly irritated. These become pigmented and are of a buff or brown colour. It is a very chronic eruption beginning in early infancy, and continuing on and off until puberty, when the eruption often tends to disappear. The little nodules or tumours are composed of masses of mast cells, which are chiefly to be seen around the blood vessels.

TREATMENT.—In acute cases due to unsuitable ingesta, a purge is indicated, and sometimes an emetic is desirable. Keep the patient on milk diet for a few days. In the more chronic cases treat any dyspepsia on ordinary lines: non-fermentable food, saline purges, and intestinal antiseptics are generally necessary. Vascular tonics (strychnine or ichthyol in pill or tabloid form) may be tried. Salicylate of soda or salol is often of service internally. Local treatment is required for the itching. Alkaline baths and dilute carbolic lotion (1 in 100) are useful.

PEMPHIGUS.

Pemphigus is a rare disease, and is characterised by the presence of *bullæ*, which appear in successive crops on previously healthy skin. It is due to internal causes, rather than to influences acting from without. In *P. vulgaris* the *bullæ* gradually increase in size and may become an inch or more in diameter. They contain a clear serum, which may become turbid later. There is no large excess of eosinophile cells in the serum from the *bullæ*, as in dermatitis herpetiformis and the rare forms of pemphigus known as pemphigus foliaceus and vegetans. The *bullæ* are most abundant on the limbs and are often symmetrical, but may affect the whole body: the mucous membranes of conjunctiva, mouth, or vagina are sometimes attacked. The disease may be acute and associated with fever and general symptoms, and may soon prove fatal. The chronic form is more common. As a rule the constitutional disturbance is slight; but sometimes death occurs from exhaustion. The pathology of the condition is unknown, but there is reason to think that it may be a specific infective disease.

TREATMENT.—Arsenic is the most efficacious remedy, but it should be pushed. In very acute cases it may be replaced by opium or quinine. Rest and nourishing diet are essential. The local treatment is less important: the *bullæ* should be protected from injury and infection from without. Dilute lotions or ointments containing liq. carbonis detergens or carbolic acid are useful.

DERMATITIS HERPETIFORMIS.

DERMATITIS HERPETIFORMIS is a polymorphic eruption with erythematous vesicular and bullous lesions, which itch violently and tend to recur in crops.

In its bullous form it closely resembles pemphigus but bullæ and vesicles occur on erythematous areas as well as on the healthy skin. The general health is also—unlike pemphigus—usually well maintained. The fluid in the vesicles and bullæ shows a very large number of eosinophile cells and there is always an excess of these cells in the blood.

HYDROA GRAVIDARUM is *Dermatitis herpetiformis* occurring during pregnancy. It usually starts between the third and sixth month, and may recur in each pregnancy or sometimes after delivery. Its treatment is the same as for *dermatitis herpetiformis*.

The treatment of *dermatitis herpetiformis* should be conducted on similar lines to that for pemphigus. Arsenic is one of the most useful drugs, and sulphur baths and ointments are valuable. When the bullæ are large, dusting powders are more useful.

HERPES.

HERPES ZOSTER or SHINGLES consists of groups of vesicles occurring on an erythematous base. The distribution of these vesicles is characteristic, for they occur in an area of skin supplied by a posterior nerve-root or of its morphological equivalent (in the case of the cranial nerves). The disease is commonest in childhood, but may occur at any age, and commences suddenly with febrile symptoms, and the eruption is accompanied or preceded by considerable pain and hyperæsthesia. The vesicles contain clear fluid, which may become turbid or blood-stained. They heal in about a month, and may leave permanent scars or be followed by severe neuralgia. The disease is almost always unilateral, and (on the trunk) the groups tend to form a band two or three inches wide running half round the body.

Pathologically, *H. zoster* depends upon lesion of the ganglion of the posterior roots of the spinal nerve corresponding to the affected area. Second attacks are extremely rare, indicating an acquired immunity. A similar eruption is sometimes induced by the administration of arsenic.

Treatment.—The vesicles should be protected by means of cotton-wool dusted with zinc and starch. Tonics should be given, and anodynes may be necessary for the relief of pain.

H. FEBRILIS has no relation to the above, and occurs round the mouth or cheeks in patients suffering from dyspepsia, catarrhal affections, sore throat, or acute fevers—especially pneumonia. Small vesicles appear, which soon rupture, leaving excoriations which become covered with a scab. Recovery soon takes place if the area is kept clean, and a simple antiseptic ointment applied.

H. GENITALIS is a similar affection of the genital regions, especially of the prepuce and glans penis. As to treatment—cleanliness, antiseptic lotions, and dry dressings are indicated.

ECZEMA.

ECZEMA is difficult to define. It is a superficial dermatitis which is due to internal causes, or to the action of external irritants on a susceptible skin. A marked characteristic is the presence of a serous exudation, which may be very abundant or extremely scanty; it stiffens linen wetted with it.

Eczema usually passes through the stages of hyperæmia, exudation (with the formation of papules or vesicles, or both), and desquamation, the epidermic scales being glued together by secretion to form crusts. The vesicles may become pustular, or the inflammation may spread to the deeper layers of the corium, causing induration in chronic

cases. The best classification is that which is based on the predominance of one or other of these stages. If hyperæmia predominates it is called *E. erythematosum*; *E. papulosum*, *vesiculosum*, *squamosum*, and *pustulosum* are self-explanatory. *E. rubrum* is most common on the legs of elderly persons, and in it the surface of the skin is red and raw, and yields much secretion. *E. rimosum* is a chronic form which occurs on the hands, especially when often wetted. It is associated with much induration, and with deep cracks or rhagades. These forms represent different phases of eczema rather than independent varieties. In any case eczema tends to become chronic, though it often begins acutely. Eczema in children is apt to be more pustular than in adults, and is especially common on the face and scalp.

Eczema has a special tendency to make its first appearance on the flexor surfaces, and at the junction of skin and mucous membranes, though it may afterwards extend to any region. The chief symptom is itching; constitutional symptoms are rarely prominent, and may be entirely absent.

The pathology of the disease is not clear. Eczema has several points of similarity to catarrh of mucous membranes, and is often spoken of as a catarrhal inflammation of the skin. It is not due primarily to the action of bacteria, though these may invade the affected part and modify the disease. Many cases are associated with gout, chronic nephritis, asthma, or dyspepsia, and may be due to the action of a toxin on the skin. Some cases appear to have a nervous origin.

The polymorphism of eczema often renders the diagnosis somewhat difficult. The history of the onset of the disease, the distribution of the eruption, and the presence or absence of serous exudation are important points. The whole eruption must be examined carefully, and an attempt

made to find vesicles, which, if present, are very characteristic. Sources of external irritation should be sought for. Lastly, there should always be a careful inquiry into the *general health*, as this is of great importance in treatment.

TREATMENT.—In acute cases the patient should be put on a bland diet, and a saline purge is generally desirable. Vinum antimoniale (℥ x. three times a day) is of some value. The local treatment consists in the avoidance of irritation, and the application of mild sedative, astringent, and antiseptic remedies, best in the form of lotions or powders. No soap should be used, and the part should be cleansed daily with milk, diluted gruel, or olive oil. The lotions which are most generally useful are given in the appendix. Lassar's paste or diachylon ointment is also available in this stage.

In chronic cases arsenic is of some value, and is best given in liquid form. At this stage ointments are the best external applications, and should contain tar, pyrogallie acid, salicylic acid, ichthyol, or sulphur. All these substances have a tendency to cause irritation when used in too great strength: but when diluted, act beneficially as stimulants to the skin. The scales in dry eczema should be removed by washing with soap containing coal-tar or sulphur, or with a dilute solution of an alkali.

IMPETIGO CONTAGIOSA.

IMPETIGO is very common in badly nourished children. It occurs in groups of vesicles on an inflamed base, which rapidly become pustular; the pustules dry up into scabs which ultimately come off, leaving a pink mark without scar. In cachectic children, or as a result of picking, ulceration occasionally takes place under the scabs. This variety of impetigo contagiosa is often called *ecthyma*.

The exposed portions, especially the face, are usually attacked first, but other regions may become infected subsequently, usually by infection carried by the fingers. The lymphatic glands appropriate to the affected areas of skin often become enlarged. The bullous impetigo of young infants, sometimes called *pemphigus neonatorum* is a serious form of the disease and may end fatally.

Impetigo contagiosa is due to the streptococcus pyogenes. The treatment consists in removing the crusts by poulticing, or by bathing with mild antiseptic lotions, and the application of an antiseptic ointment, a weak ointment of ammoniated mercury being especially useful. Good food and tonics are indicated.

PSORIASIS.

PSORIASIS is a chronic affection which attacks both sexes indiscriminately, occurring at any age, and is liable to run in the same family. It is characterised by red scaly papules, which tend to enlarge and form areas of variable size, covered with thin dry silvery scales. It is particularly liable to affect the extensor surfaces of the limbs, especially the points of the elbows and knees. The scalp is also frequently attacked. It also occurs on the trunk in the form of scattered scaly papules (*P. guttata*), or in large scaly areas or in ring-shaped lesions due to clearing up of the central parts of rounded areas. When the scales are roughly removed, bleeding commonly occurs, due to injury of the subjacent papillæ. There is no itching except in acute forms, and no constitutional disturbance. Nor is there induration of the skin, except to a moderate degree in old patches on the legs. Some pigmentation of the skin may be left on the disappearance of very chronic patches.

TREATMENT.—Arsenic is of value in selected chronic cases

and should be given in the form of Fowler's solution (℥ iij. increased to ℥ x. or ℥ xv.) thrice daily after meals. Some cases do well on salicylate of soda, thyroid extract, or large doses of iodide of potassium. The external treatment is more important. The scales should be removed by an alkaline lotion in acute cases, or by thorough washing with soap and water when the disease is more chronic. After this, ointments must be rubbed thoroughly into the skin. In acute cases Lassar's paste may be used; in chronic cases more stimulating preparations are necessary, and those containing chrysarobin and tar are most efficacious. Chrysarobin should be used with care, as it may set up a severe dermatitis and if used near the eyes may cause conjunctivitis.

LICHEN PLANUS.

LICHEN PLANUS is characterised by the development of crops of small, hard, flat-topped papules which have an *angular* or polygonal shape. The surfaces of these papules have a peculiar burnished or shiny appearance, and show fine lines or points (Wickham's *striae*) and are sometimes depressed in the centre; they have a red colour when they make their first appearance, but afterwards become violet, and in chronic cases brown. These papules occur in groups, the component lesions of which may become confluent, and in this case the diagnosis may be difficult; but a few typical papules will usually be found near the edge of the eruption, and when this is the case, the nature of the disease is evident. *L. planus* usually attacks the flexor surfaces of the wrists and forearms and the inner aspects of the knees, but it may occur anywhere except upon the scalp; it is usually symmetrical. The mucous membranes are not uncommonly attacked, and on the inner aspect of the cheeks small white raised patches or

papules may often be seen. Itching is sometimes very severe.

TREATMENT.—The drugs which have most influence are arsenic, antimony, and perchloride of mercury, and these should be given in full doses. The most useful external applications contain tar, perchloride of mercury, or carbolic acid; salicylic acid is often necessary in the treatment of chronic confluent patches.

PITYRIASIS RUBRA.

The term pityriasis denotes a *scaly* affection, and is applied to several diseases, of which *P. rubra* (*dermatitis exfoliativa*) is the most important (in spite of its rarity) in that it is one of the few skin diseases which may terminate fatally. It sometimes arises as a primary affection, but it is more common for it to develop after other skin diseases (especially psoriasis and eczema) which have been treated with irritant applications for a prolonged period. The skin of large areas or of the whole body has a vivid red colour and desquamates with great rapidity; the epidermis is shed in thin flakes in such abundance that the bed may become filled with them in the course of a few hours. Yet there is little or no thickening of the skin, and itching is commonly absent. The prognosis should be cautious, as a fatal result is of frequent occurrence, either rapidly with typhoidal symptoms, or slowly from exhaustion.

Treatment.—One of the most important indications is to keep the patient warm, as the subjects of pityriasis rubra are always extremely sensitive to cold, and chills probably aggravate the disease.

The patient's strength should be supported by nourishing and digestible food, and the internal administration of tonics or cod-liver oil. Arsenic should not be used in the early stage, and is not of much use at any time. The

value of external applications is doubtful, but dilute astringent and antiseptic lotions or ointments may be tried.

DISEASES OF THE HAIR FOLLICLES.

Inflammation of the hair follicles is due to local infection of the follicle or its sebaceous gland with micro-organisms, commonly the *staphylococcus aureus*.

FURUNCLES—BOILS.

In boils, the cocci enter deeply into the hair follicle and sebaceous gland, and set up inflammation which extends into and involves the surrounding tissue. The centre of this area suppurates or forms a slough, over which the skin breaks down, and the slough comes away. A large and severe boil, with a flat indurated centre, which involves several hair follicles and tends to break down and discharge pus through cribriform openings, is called a carbuncle.

SYCOSIS (COCOGENIC).

In this affection the follicles of the beard are affected. A deep-seated folliculitis is thus set up, and the beard area becomes covered with pustules, each of which is perforated by a hair. The hairs are soon loosened, and may be easily withdrawn. The follicular papilla is seldom destroyed, so that the hair soon grows again when the inflammation has subsided. Sycosis may be confounded with eczema of the beard region, from which it may be distinguished by the fact that the skin between the follicles is but slightly inflamed; or with *tinea barbæ*, when due to the *ectothrix trichophyton*, in which case there is a brawny infiltration of the deeper portions of the skin.

Treatment of Boils and Sycosis.—Obstinate cases of both

may be advantageously treated by inoculations of vaccines prepared from dead staphylococci. The vaccine can be made from the patient's own pus or a stock vaccine may be used. The initial dose may be 75 to 100 millions, and doses are usually given about every ten days until dosages of 250 to 500 millions have been reached. A boil which has once fairly started cannot be cut short. Painful tension may be relieved by incision, and the surrounding skin should be frequently washed with an antiseptic solution to prevent inoculation of adjacent follicles. The general health requires close attention. Fresh brewer's yeast—an old-fashioned remedy—is certainly of use in some cases of boils. It has been found that it raises the opsonic index to staphylococci. Arsenic in very small doses as recommended by Duhring is also sometimes useful.

In sycosis all affected hairs should be extracted, followed by inunction with oleate of mercury (10 per cent.). Epilation by X-rays is a very useful treatment, as large areas can be treated without pain. A full pastille dose is given, and the hair falls out about three weeks later.

ACNE.

ACNE is a common affection of the hair follicles usually beginning about puberty. The initial lesion takes the form of what are known as *comedones*. A comedo consists of a little mass of horny cells and fat which plugs the neck of the follicle, forming a little prominence on the surface, surmounted by a black point, popularly known as a "black-head" (*A. punctata*). It can readily be squeezed out by pressure. Comedones are most common on the face and shoulders. They are probably due to local bacillary infection, and the organism most commonly found is the microbacillus of seborrhœa (Sabourand) or the bacillus acne. They are apt to become inflamed owing to secondary

infection with pyogenic cocci, forming red papules which suppurate (*A. vulgaris*). As a rule they rupture, discharge their contents, and soon heal ; but they frequently become indolent and are attended with considerable infiltration of adjacent tissue (*A. indurata*). In this case they are slow to heal, and are often followed by disfiguring scars.

Treatment.—The affected part should be thoroughly washed with hot water and soft soap, or soap containing sulphur, tar, or ichthyol, and such comedones as will come out easily should be expressed with a watch key, or other suitable instrument. In acne vulgaris, pustules should be incised, and dressed with calamine and sulphur lotions. In severe cases of the indurated type, mild applications of X-rays will often prove beneficial. If the scalp is affected with seborrhœa, it should be well washed, and anointed with sulphur and salicylic ointment.

Internal treatment is often of value ; dyspepsia, constipation, or anæmia should be treated ; and yeast sometimes does good in the pustular cases. In chronic and indurated cases vaccines are sometimes most useful, but examination should first be made to determine the organisms which are responsible.

ALOPECIA AREATA.

ALOPECIA AREATA is a disease of the scalp in which the hairs over a circumscribed and approximately circular patch are rapidly shed ; the bald patch thus left is white and polished and its surface is depressed as the result of atrophy. At the margin of the patch short atrophied hairs are found, which come out easily. It extends gradually, and other patches may form. The disease is very chronic, but it almost always recovers after a longer or shorter period, except in the severer forms.

The pathology of the disease is not definitely established,

and it is probable that while some forms are due to parasitic causes, others are of nervous origin.

The disease is sometimes confounded with ringworm, but in this affection the patches are more likely to be scaly, the hairs at the margin are broken and thicker, and on extraction the characteristic fungus can be readily identified by microscopic examination. Ringworm can also be proved to be present by making cultures with the affected hairs on maltose agar.

Treatment.—Locally, stimulating and antiseptic lotions are indicated. Blistering may be cautiously used in some cases. The general health requires attention.

DISEASES OF THE SWEAT-GLANDS.

SUDAMINA are due to the profuse secretion of sweat by glands, the orifices of which are obstructed, appearing as small vesicles with clear contents in the stratum corneum, which sometimes become slightly inflamed. The blocking of the ducts may occur as a result of previous diminution of the secretion of sweat, and is facilitated by hyperæmia of the skin; hence sudamina frequently occur with the rashes of the exanthemata. They are common in all diseases which are associated with a high temperature and profuse perspiration, especially in pneumonia, typhoid fever, and acute rheumatism, and may occur in perfect health during hot weather or after violent exercise. No treatment is necessary, for the affection is transitory and causes no inconvenience.

MILIARIA also occur in patients who sweat profusely, but they differ from sudamina in that the vesicles which are formed are due to distension of sweat-ducts in the deeper layers of the epidermis. The lesion makes its appearance in the form of a crop of minute papules of a bright red colour. Vesicles are formed on the apices of some or all

of these papules, and contrast markedly in colour with their backgrounds. They occasionally become pustular, and staphylococci are found in the vesicles and pustules.

STROPHULUS or "red gum" is a common affection in children, especially if they are too thickly clad, and is allied to miliaria. *Prickly heat* is also a similar disease, and is common in the tropics; it is accompanied or preceded by profuse itching.

Treatment.—A dilute antiseptic and sedative lotion of carbolic acid or creolin is all that is required.

SEBORRHŒA AND ITS ALLIES.

SEBORRHŒA CAPITIS (scurf or dandruff) occurs in two forms, the dry and the oily (*S. sicca* and *oleosa*). It is of little importance in itself—except as a common cause of baldness—but it is of great importance in that it causes or modifies eruptions in other parts of the body; and the successful treatment of these affections depends on the recognition and treatment of the primary cause in the scalp. The pathology of the condition is not definitely known, but there is reason to think that it is due to a micro-organism and that the excess of fat which is so characteristic comes from the sweat rather than the sebaceous glands.

Seborrhœa corporis is probably the same disease affecting the body, and seems to be due to infection from the scales which dust down from the head. It is very common, especially in those who wear flannel next to the skin, who sweat freely, and who wash seldom. *Seborrhœa corporis* nearly always develops first in the middle line of the trunk, the earliest lesions being seen over the sternum and on the back between the shoulders. The eruption begins as a red papule, which is covered by yellowish scales; these contain fat, and feel somewhat like pastry when rolled

between the fingers. These papules extend laterally, and form patches of considerable size which may clear in the centre. The disease was called lichen circinatus by old authors.

Seborrhæic eczema is a more serious affection, follicular in its origin, and spreading serpigiously. It is undoubtedly of a parasitic nature and staphylococcal infection is frequently present. It is most common on the scalp, but it often extends to the face, and no part of the body is exempt.

TREATMENT.—The parts should be thoroughly washed with soap and water, followed by the use of salicylic and sulphur ointment—provided there is not much associated inflammation. Mild tar lotions are also useful.

LUPUS.

LUPUS VULGARIS is the commonest form of tuberculosis of the skin. It is caused by the presence of tubercular bacilli in the corium; they are present in very scanty numbers, and are embedded in small masses of granulation-tissue. These nodules are at first of small size and are red in colour; they grow gradually larger, become translucent, and assume a reddish-yellow colour, and are then often compared to small masses of apple-jelly. The epidermis is raised by these nodules, and frequently desquamates; and the corium around the granulations becomes inflamed and thickened by the formation of new fibrous tissue. The disease is very chronic, and the lesions gradually spread by peripheral expansion due to the formation of fresh nodules, and the affected area may ultimately become very large.

The lesion may ulcerate, or may undergo cicatrisation without previous ulceration. The lupoid ulcer has a raised edge, in which the semi-translucent nodules may

still be seen ; and its base is usually covered with scabs, and secretes a small amount of discharge ; or it may be covered with exuberant granulations. It heals very gradually, leaving a thin and bluish-white cicatrix, in which the original disease often reappears.

L. vulgaris usually begins before puberty and persists indefinitely unless properly treated. It is commonest on the face, especially on the nose and cheeks, and rarely arises on the scalp, but may affect *any* region of the skin. It may spread to mucous membranes, especially those of the mouth and larynx, or may occur primarily in these situations. *Lupus vulgaris* differs from *lupus erythematosus* in that it is very rarely symmetrical.

The diagnosis is often facilitated by removing any scales which may be present, and then rendering the skin anæmic by gentle pressure with a piece of glass ; in this way the characteristic nodules are easily seen. Old tuberculin causes a local reaction in the affected tissues and inflammation of the lupus patches may follow the inunction of a 5 to 10 per cent. tuberculin ointment. Patients with *lupus vulgaris* react to Von Pirquet's, Moro's and Calmette's tests. *Lupus* may be confounded with *L. erythematosus*, which is usually symmetrical and never ulcerates ; with tertiary syphilides, which run a more rapid course and are cured by appropriate treatment ; or with rodent ulcer, which commences later in life and extends more slowly.

TREATMENT.—Surgical measures are much less used than formerly. The Finsen treatment by light-rays yields good results when the mucous membranes are not involved. The X-rays are also valuable when carefully used. Short exposures are required from time to time. If a few nodules are left after these methods, they may be dealt with by strong carbolic acid applied on a wooden point. Tuberculin injections have been recommended, but are seldom em-

ployed. New tuberculin T. R., even now that its action can be controlled by opsonic observations, has proved disappointing, especially in the dry nodular variety of lupus. In slight cases there is no need for any of the above methods—and the nodules may be destroyed by the galvano-cautery, or acid nitrate of mercury. Salicylic acid, in the form of Unna's salicylic-creosote plaster, is often useful.

L. ERYTHEMATOSUS is an inflammatory erythematous disease which occurs in two varieties, (1) the localised, which is far the commoner, and (2) the disseminated. In the localised form the disease appears as an erythematous patch which gradually extends peripherally, while the central part may undergo slow atrophic and cicatricial changes without previous ulceration. The erythematous edge may contain numerous dilated sebaceous follicles and be covered by thin scales, with conical epidermic plugs on their under surfaces which dip into the distended follicles below. The disease varies very much in its severity, the milder superficial forms closely resembling erythema multiforme.

It is most common in early middle life and runs a very chronic course. The central parts of the face are most often attacked first; it then spreads laterally, producing a "butterfly-shaped" patch which extends over the nose (representing the body of the butterfly) and the adjacent portion of the cheeks. It may attack the ears or the scalp (in the latter case a permanent bald patch is left) or the mucous membranes of the lips and mouth. Lupus erythematosus rarely affects the hands, and when it does so, it closely resembles chilblains.

A *disseminated form* may occur as a complication of the more chronic fixed type, or it may run an acute course from the start. Dull red pink or purple red areas coalesce into the "butterfly" shape on the face, with

very little scaling if any, and similar areas occur on the trunk and limbs. The patient is often gravely ill with a high temperature, and albuminuria and the more severe cases of this variety may go on to a fatal issue.

The pathology of the disease is still obscure, but as it often occurs in patients who have tubercular lesions in other parts of the body it is believed to be due to the action of the toxins of the tubercle bacillus on the skin. Gougerot has produced tuberculosis in guinea-pigs by inoculation of diseased material, and quite recently by improved methods tubercle bacilli have been found in the skin. Some think that the disease may be due to other toxins besides those produced by the tubercle bacillus.

The *treatment* is unsatisfactory. It is well to avoid any strong application, until one sees what can be done by strict attention to the general health. A simple calamine lotion will suffice. Later on, collodion with 2 per cent. salicylic acid may be painted on, and in some cases linear scarification proves useful.

RINGWORM.

RINGWORM is due to the invasion of the skin or hair follicles by certain fungi, of which two species are of common occurrence in this country. *Microsporon audouini*, the small-spored fungus, is the cause of the great majority of cases of ringworm of the scalp: it may be recognised microscopically by the fact that the spores have an indefinite mosaic-like arrangement. *Trichophyton megalo-
sporon*, the large-spored fungus (of which there are several varieties), is a rare cause of ringworm of the scalp, but is common on other parts of the body: it may be recognised by the fact that the spores are arranged in chains or rosaries.

Ringworm is very infectious, and may be acquired from

cases occurring in man or in the lower animals, especially from horses, cats, and dogs. Children who suffer from it must be isolated; and brushes, combs, and towels should be kept for their sole use.

The diagnosis is established by the discovery of the fungus on microscopic examination; this is facilitated by the fact that the spores and mycellum resist the action of caustic potash, while the keratin of the hair or epidermic scales is rendered translucent.

RINGWORM OF THE SCALP (*T. tonsurans*) is common in children and very rare in adults. It begins in the form of a small rounded scaly area over which the hair is more or less lost. On examination broken hair-stumps can be seen, often surrounded by little scaly sheaths. The patch spreads by marginal extension, and several areas may become affected. If neglected, the disease runs a chronic course. A much less common form, caused by a variety of the large spored fungus (endothrix), is characterised by widely scattered groups of a few hairs each, broken off close to the scalp (black-dot ringworm), and it is very easily overlooked.

Kerion is the term applied to an inflammatory condition of ringworm of the scalp in which the patch becomes red, swollen, and boggy to the touch; and the hair follicles exude a thick purulent secretion. A closely similar condition is met with on the arms or back of the hands as the result of an infection from animals. It is due to the large-spored fungus (*Ectothrix* variety).

RINGWORM OF THE BODY (*T. circinata*) may be due to the large- or small-spored fungus, and presents circular patches with a bright red colour and scaly surface: they extend rapidly and often clear in the centre, thus forming narrow red rings. In moist regions, especially in the groin, the inflammatory symptoms are more marked—a condition sometimes known as *eczema marginatum*, which has been

found to be due to a special variety of fungus called the epidermophyton inguinale (Sabourand). This form of the disease is highly infectious and spreads rapidly in schools and other large institutions.

RINGWORM OF THE BEARD (*T. barbæ* or *Tinea sycosis*) may occur in the same form as *T. circinata*, but in the more common form of the disease the hair follicles are inflamed and exude pus. It closely resembles coccogenic sycosis, but may be recognised by the presence of hard inflammatory nodules situated deep in the skin. This condition is similar in nature to kerion, and the ectothrix is usually the variety of fungus present. In the dry scaly form of ringworm of the beard, endothrix trichophyton may be found.

RINGWORM OF THE NAILS is rare, and usually occurs in those who have ringworm in other parts of the body. The nail becomes dull and lustreless, and readily breaks. The diagnosis can only be made with certainty by a microscopic examination of scrapings.

TREATMENT.—The fungus is easily destroyed by the application of parasitocides, but when hairy regions are affected it is very difficult to get any substance to penetrate to the recesses of the follicles. The hair of the affected area and of a wide margin round it should be clipped or shaved, and the skin washed thoroughly with soft soap and hot water, and then with ether or turpentine; it should then be allowed to dry, after which antiseptic lotions or ointments may be applied. Epilation by the X-rays is now frequently practised in ringworm of the beard, after which parasitocides can be used with greater advantage.

FAVUS.

FAVUS is most commonly due to the growth of a fungus called the *Achorion Schönleinii* and closely allied to the

Trichophyton. The scalp is the usual seat, but it may affect the glabrous portions of the skin. It presents sulphur-yellow umbilicated crusts or scutula, each about a third of an inch in diameter: these often coalesce to form yellow masses, and have a peculiar mouse-like odour. A serious cicatricial atrophy of the scalp with permanent baldness may follow favus of the scalp.

TREATMENT.—The scutula should be removed as completely as possible by soaking in carbolised oil, and the affected hairs pulled out carefully with a pair of forceps. When this has been done, antiseptic or stimulating applications must be employed. Epilation with the X-rays is again useful, as in ringworm, in expediting the eradication of the disease.

PITYRIASIS VERSICOLOR.

P. VERSICOLOR is due to a fungus known as *Microsporon furfur*, and occurs in the form of small yellowish branny patches, in which the fungus is easily demonstrated. It is most common on the chest, especially in those who change their clothing but seldom, and seems to affect phthisical patients with special frequency. It is cured by cleanliness and the use of an antiseptic lotion.

DISEASES DUE TO ANIMAL PARASITES.

SCABIES is due to infection with the *Acarus scabiei*, the impregnated female of which burrows obliquely into the horny layers of the skin (depositing her eggs as she does so) and causes a condition closely resembling eczema. A small vesicle shows the commencement of the burrow, the site of which is marked by a small rough ridge: the acarus lies at the bottom of this burrow, and may sometimes be removed with the point of a needle and identified microscopically.

The primary lesion in scabies is the vesicle, but secondary infection with pyogenic cocci often leads to the formation of pustules, and the inflammation may spread to the corium and cause much increase of fibrous tissue, or fissures may form. This disease causes much itching, which is characteristically worse at night, and scratch-marks are very common.

Scabies is most common in the thin skin between the fingers, on the external genitals, and on the anterior fold of the axilla. The distribution of the lesions affords help in the diagnosis of this disease from eczema. The fact that the scabies is infectious, and that two persons who occupy the same bed, or come into close contact in other ways, may be affected simultaneously, frequently afford a clue as to the nature of the disease.

Treatment.—This consists of thorough scouring of the skin with soap (preferably soft soap) and hot water, in order to open up the burrows, followed by free inunction with an ointment containing sulphur or other parasiticide agent. The underclothing must be disinfected by boiling, and the outer garments by baking, before being worn again, or they may lead to a renewed infection.

PHTHEIRIASIS.—*Pediculi* suck blood through a proboscis which is inserted into a sweat-duct, and in doing so cause minute subcuticular hæmorrhages: in susceptible persons a wheal may develop round the puncture. There is much itching, and the scratch-marks may become infected with pyogenic bacteria, causing a pustular rash.

The *pediculus capitis* affects the hairy scalp, and may be diagnosed by the presence of spindle-shaped eggs (nits) which are attached to the hairs by means of a firm cement. The parasite causes much irritation, and gives rise to a condition which closely resembles eczema; the neighbouring lymphatic glands are often enlarged.

Treatment.—In young children the hair may be cut

short and the scalp treated with white precipitate ointment. In older women the hair need not be cut. A strong lotion of carbolic acid (1 in 20) may be used for soaking the hair and scalp, care being taken to prevent irritation of the face and eyes. This lotion rapidly kills both insects and nits. Acetic acid may be used to loosen the nits whilst they are being combed out.

The *pediculus corporis* is larger than the above, and affects old persons of dirty habits. It causes much itching, and numerous scratch-marks may be seen on the back, whilst the parasites may be found under the seams of the underclothing.

Treatment.—Personal cleanliness and frequent change of the underclothing, which should be boiled before being worn again.

The *pediculus pubis* is smaller and squarer than the foregoing, and inhabits the pubic region, or, more rarely, the eyelashes.

Treatment.—Application of white precipitate ointment is usually sufficient; the pubic region should not be shaved.

DRUG ERUPTIONS.

Many substances act as irritants and cause lesions of the skin when applied externally, but these will not be considered here. The drugs which affect the skin when taken internally are of great importance, as they often lead to mistakes in diagnosis, and it is a golden rule to inquire into the nature of the medicines which are being taken by any patient who exhibits a skin disease which is anomalous in appearance or which resists appropriate treatment. It must also be remembered that certain patients have peculiar idiosyncrasies towards certain drugs, and that in them a small quantity may determine the production of a rash. These drug eruptions usually disappear spon-

taneously when the administration of the substance which causes them is stopped.

Arsenic may produce erythema, herpes, vesicles in regions in which the skin is thin, thickening of the epidermis (callosities) under which epithelioma may develop, or brown pigmentation.

Antitoxins frequently cause a scarlatiniform or urticarial eruption, which usually appears a week or more after the injection of the serum.

Atropine, or *belladonna*.—An erythematous rash resembling scarlet fever.

Bromides.—Erythematous, vesicular, or papular eruption, usually on the face or neck; or a pustular eruption resembling acne.

Copaiba.—Erythematous or purpuric eruption, attended by much itching and burning.

Iodides.—Erythema, bullæ, or hard papules, which frequently become pustular.

Quinine.—Erythema or urticaria.

Silver.—Prolonged administration may lead to a permanent bluish discoloration of the skin.

Rashes frequently follow the administration of *enemata*; they resemble those of scarlatina or measles, and may be followed by slight desquamation. They last for a day or so, and appear to be due to the absorption of fæcal material dissolved by the *enemata*.

TUMOURS.

The numerous and complicated structures which enter into the formation of the skin render possible the development therein of many forms of tumours. These are mainly of surgical interest, and little more than an enumeration of the more important varieties will be attempted.

They are best divided into the *malignant* and *benign* forms.

MALIGNANT TUMOURS.—(1) Epithelial :—

*Epithelioma.**Rodent ulcer.**Paget's disease of the nipple.*

(2) Connective tissue :—

*Sarcoma.**Melanotic sarcoma.*

BENIGN TUMOURS.—Epithelial :—

Corns and warts.

Molluscum contagiosum.—The tumours are usually multiple and consist of small firm plaques which commonly have a central depression. They may become pedunculated. The disease is contagious, and has been attributed to the action of protozoa.

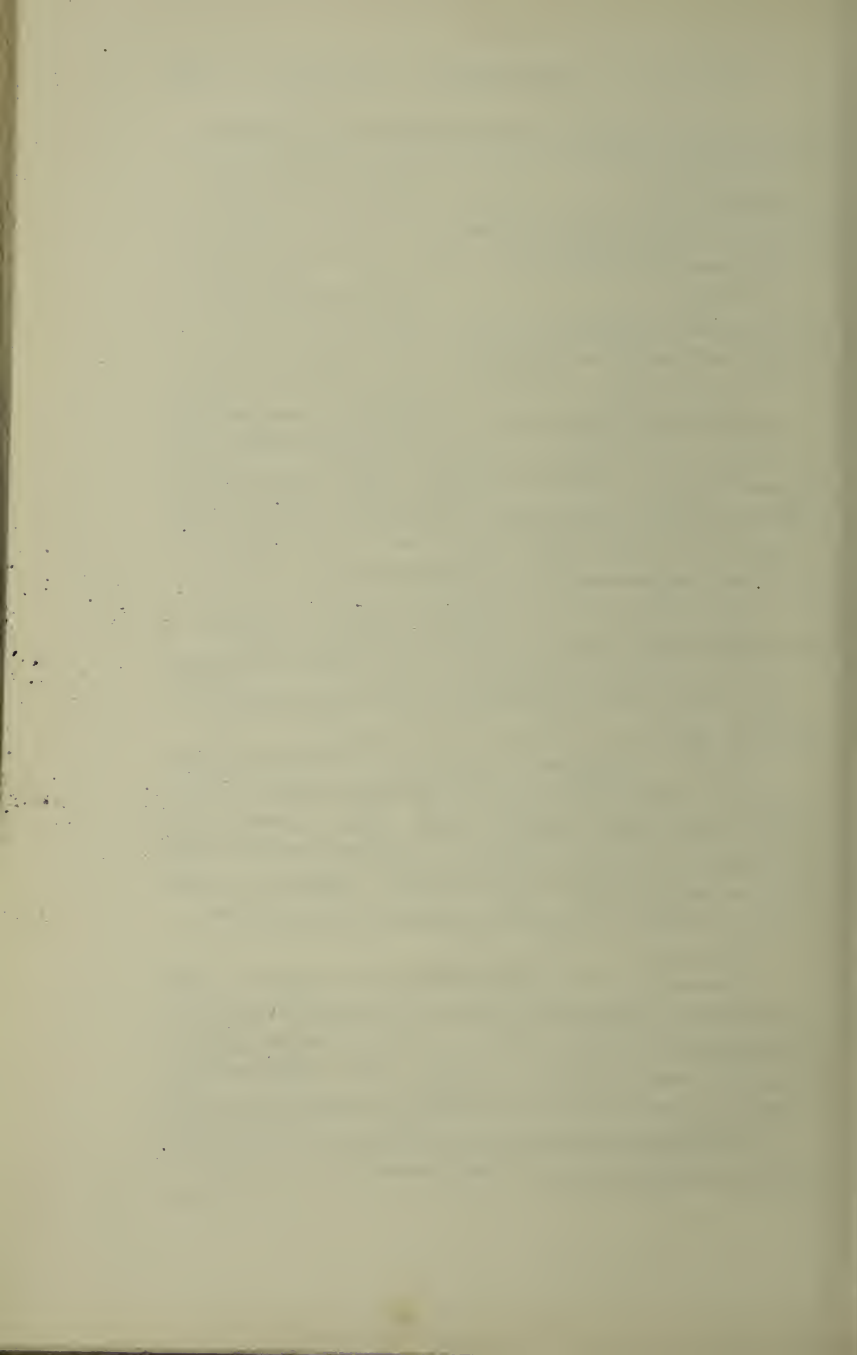
Nævi (angiomata) and *pigmented nævi.**Fibromata.*

Molluscum fibrosum.—Multiple fibrous tumours of variable size and consistency, and often accompanied by multiple fibromata of the nerves and pigmentation of the skin.

Keloid.—A progressive hypertrophy of the scar-tissue, forming hard white or pink tumours, covered by their skin and usually slightly raised. They should not be excised, as recurrence almost invariably takes place, and some cases recover spontaneously. Keloids can often be successfully removed by treatment with radium.

Neuroma.

Lipomata.—Under this heading some authors include *Xanthoma*, a disease in which small raised yellow patches develop in the skin. They are most common upon the eyelids, and in diabetic subjects. The yellow colour is due to the presence of a fat-like material, but this is probably to be regarded as an infiltration of the muscular fibres rather than as a true tumour.



APPENDIX A.

THERAPEUTIC INDEX.

NOTE.—In submitting the following therapeutic index, my intention has been merely to provide examples of prescriptions which have been found useful by myself and others, and to suggest in a general way the lines of treatment usually adopted. Any slavish adhesion to fixed formulæ is strongly to be deprecated as tending to stifle the spirit of original inquiry on the part of the practitioner, and taking the place of that which personal practice and experience alone can properly give. The suggestions made under each heading should be read in conjunction with the paragraphs in the text which relate to the same subject.

GENERAL DISEASES.

Ague.

1. R Quininæ sulph. gr. x.
Towards end of sweating stage and gr. v. every four hours for three doses.

2. R Quininæ sulphat. gr. i.
Ferri arseniat. gr. $\frac{1}{6}$ - $\frac{1}{4}$.
Glycerin. tragac. q.s.
Ft. pil. i.

Thrice daily after food. In malarial anæmia.

3. Warburg's tincture, $\overline{3}$ ss.

Give undiluted, after opening the bowels. Repeat in three hours.

Alcoholism.

1. R Ammon. carb. $\overline{3}$ i.
Table vinegar $\overline{3}$ ii.

A useful reviver from drunkenness.

2. R Liq. arsenicalis $\mathbb{M}$ i.
Tr. capsici $\mathbb{M}$ i.
Aquam ad $\overline{3}$ i.

Every hour. Useful in persistent vomiting after a debauch.

3. R Morph. bimecon. gr. $\frac{1}{4}$ - $\frac{1}{2}$.
Subcutaneous injection.

or R Hyoscinae hydrobromid.
gr. $\frac{1}{200}$ - $\frac{1}{100}$. Subcutaneous injection.

or R Sulphonal gr. xx.-xl.

or R Trional gr. xx.-xxx.

Dissolve in boiling water, or give in hot milk. Useful sedatives in delirium tremens.

4. R Liq. strychninae $\mathbb{M}$ v.
Tr. capsici $\mathbb{M}$ x.
Aquam ad $\overline{3}$ i.

Every four or six hours. To relieve chronic alcoholic depression.

5. R Tr. cinchon. co.
 Sp. ammon. arom. āā ʒss.
 Tr. capsici ℥x.
 Tr. card. co. ℥xx.
 Aquam ad ʒi.

A dose when craving for drink comes on in chronic alcoholism.

Anæmia.

1. All forms of iron.
 2. R Ferri sulph. gr. ii.
 Magnes. sulph. gr. x.-xx.
 Acidi sulph. dil. ℥x.
 Aq. menth. pip. ʒi.

Thrice daily. In anæmia with constipation.

- or R Pil. aloes et myrrh. gr. iv.
 Ext. nucis vom. gr. $\frac{1}{2}$.
 Ferri arseniat. gr. $\frac{1}{8}$.
 Ft. pil. i.

Daily after dinner, in constipation with anæmia.

3. Pil. ferri B.P. gr. v.
 One or more thrice daily in chlorosis (Blaud's pill).

4. R Potass. bromid. ʒi.
 Ferri et am. cit. ʒss.
 Sp. ammon. ar. ʒi.
 Aquam ad ʒvi.

ʒi thrice daily. In anæmia with nervousness.

5. R Liq. arsenicalis ℥iii.-v.
 Ferri et am. cit. gr. v.
 Aquam ad ʒi.

Thrice daily, after food, in pernicious anæmia.

6. R Parrish's chem. food,
 ʒss.-i.

Thrice daily. In anæmia of rapidly growing children.

Aneurism.

1. R Potass. iodidi gr. xx.-ʒi.
 Sp. ammon. arom. ℥xv.
 Infus. calumbæ ʒi.

Thrice daily, after food.

2. R Morphiæ hydrochlor. gr. i.
 Acid. hydrochlor. dil.
 Acid. hydrocyan. dil.
 āā ℥xvi.
 Syrupi scillæ ʒi.
 Aquæ ʒi. M.

One teaspoonful occasionally.

- R Syrupi scillæ ʒii.
 Tr. lavand. co. ʒii.
 Tr. opii ammon. ʒss.
 Syrupi simp. ʒi.
 Aq. menth. pip. ʒii.

A teaspoonful occasionally. Useful for aneurismal cough.

3. R Ext. belladonnæ ʒi.
 Ext. opii ʒi.
 Sp. vini rect. ʒss.
 Glycerini ʒss.

Useful external application for aneurismal pains.

Angina Pectoris.

1. R Sp. ætheris co. ʒss.-i.
 Liq. opii sed. ℥v.
 Aq. camph. ad ʒi.

Occasionally in water as required.

2. R Tr. ferri perchlor. ℥x.
 Liq. strychninæ ℥iii.
 Liq. arsenici hydroch.
 ℥iii.
 Aq. chloroformi ad ʒi.

Thrice daily for cardiac pain.

3. R Tr. digitalis.
 Tr. strophanthi āā ℥v.
 Liq. trinitrin. ℥i.-ii.
 Tr. cardam. co. ℥xx.
 Aquam ad ʒss.

Thrice daily. Useful between the attacks.

4. R Trinitrin. gr. $\frac{1}{100}$.
 In tablet form. One or more as required when in pain.

5. Nitrite of amyl.
 Four or five drops to be inhaled from a handkerchief during paroxysm.

Asthma.

During the paroxysm :

1. The *inhalation* of the fumes of burning nitre paper, or of stramonium cigarettes.
2. R Potass. nitrat. $\bar{z}$ ss.
Pulv. anisi fruct. $\bar{z}$ ss.
Pulv. stramon. fol. $\bar{z}$ i.
Ignite and inhale smoke.
3. Inhalation of nitrite of amyl.
4. Subcutaneous injection of morphia (gr. $\frac{1}{6}$) with atropia (gr. $\frac{1}{16}$). Use with caution.
5. R Tr. lobeliæ æth. $\mathbb{M}$ xv.
Vin. ipecac. $\mathbb{M}$ x.
Aquam ad $\bar{z}$ ss.

Every half-hour for two or three doses ; then every 1 to 2 hours.

6. Spraying the nostrils with sol. of adrenalin chloride (1 in 2,000).
7. R Potass. iodid. gr. v.-x.
Ext. stramonii gr. $\frac{1}{4}$.
Sp. ammon. ar. $\mathbb{M}$ xx.
Aq. chloroformi ad $\bar{z}$ i.

Twice or three times a day, during the interval between the paroxysms.

8. R Quininæ sulph. gr. i.
Acidi arseniosi.
Strychninæ sulph.
aa gr. $\frac{1}{30}$.
Glycer. tragac. q.s.
Ft. pil. i

Three times a day. In chronic bronchitic asthma.

Bed-sores.

1. Wash threatened part with soap and water. Dry carefully. Dab over with lotion of equal parts of spirits of wine and liq. hydrarg. perchlor. ; when dry, dust over with lycopodium pow-

der, and protect from pressure.

2. If skin remains red, paint from time to time with flexible collodion.
3. For open bed-sore, remove direct pressure, and dress with iodoform, or equal parts of chloretone and boric acid.

Bright's Disease.**ACUTE.**

1. Milk diet. Free supply of watery drinks.
2. Hot fomentations, or mustard poultices over the loins.
3. Hot air or vapour baths, or wet-packs.
4. Hypodermic injections of pilocarpin (gr. $\frac{1}{16}$). Not suitable for children.
5. Free purgation with compound jalap powder, or sulphate of magnesia.
6. R Liq. ammoniæ acetat. $\bar{z}$ i.
Sp. ætheris nitrosi $\mathbb{M}$ xx.
Aq. ad $\bar{z}$ i.

Every four hours, with warm diluent drink.

7. R Potass. citrat. gr. xv.
Infusi digitalis $\bar{z}$ i.
Infusi buchû ad $\bar{z}$ i.

Every four hours. A useful diuretic.

8. R Tr. ferri perchlor. $\mathbb{M}$ x.
Liq. ammon. acet. $\bar{z}$ i.
Aq. chloroformi ad $\bar{z}$ i.

Thrice daily. A good tonic during convalescence.

CHRONIC.

1. Diet, diaphoretics, and purgatives as in the acute form.
2. R Sodii benzoat. gr. x.
Infusi digitalis $\bar{z}$ ii.
Decoct. scoparii $\bar{z}$ i. M,

Thrice daily. Good general diuretic.

3. R Potass. acetat. gr. xx.
Caffeini citrat. gr. iii.-v.
Tr. strophanthi ℥vi.
Aquam ad ʒi.

Thrice daily, as a diuretic.

4. *Liq. trinitrin*. ℥i. every half-hour for six or eight doses, then every two or three hours. Useful diuretic in gouty cases.

5. R Tr. ferri perchlor. ℥x.
Liq. arsenici hydroch. ℥iii.
Aq. chloroformi ʒi.

Thrice daily.

- or R Tr. ferri perchlor. ℥x.
Liq. ammon. acetat. ʒi.
Aq. chloroformi ad ʒi.

In water, thrice daily.

Bronchitis.

ACUTE.

1. R Vin. antimon. ℥x.-xv.
Vin. ipecac. ℥x.-xv.
Liq. morph. hyd. ℥x.-xv.
Aq. camph. ad ʒi.

Every four or six hours. Relaxing expectorant in early stages. Apomorphia, gr. $\frac{1}{10}$ - $\frac{1}{8}$ may sometimes replace the vin. ipecac. and vin. antimon. with advantage.

2. R Vin. ipecac. ℥v.
Tr. camph. co. ℥v.
Liq. ammon. acet. ℥xx.
Syrup. et aq. ad ʒii.

Every three or four hours. Useful relaxing expectorant for children.

3. R Ammon. carb. gr. ii.-v.
Ammon. chlorid. gr. v.
Sp. chloroformi ℥x.
Syr. tolutani ʒss.
Aquam ad ʒi.

Thrice daily. Useful stimulant expectorant in later stages.

4. R Potass. iodid. gr. ii.
Sodii bicarb. gr. x.
Tr. nucis vom. ℥v.-x.
Aq. menth. pip. ad ʒi.
Thrice daily in later stages.

5. R Ammon. carb. gr. v.
Tr. scillæ ℥x.
Sp. chloroformi ℥x.
Inf. cinchonæ ad ʒi.

Thrice daily. In capillary bronchitis in the aged.

Also alcohol in warm drinks, and hypodermic injections of strychnia.

CHRONIC.

1. R Ammon. carb. gr. v.
Tr. camph. co. ℥xx.
Infusi senegæ ʒss.

In water, three or four times daily.

2. R Apomorphiæ gr. $\frac{1}{8}$.
(Heroin. hydrochlor. gr. $\frac{1}{12}$).
Aq. camphoræ ʒss.

Every four hours. An excellent expectorant in irritative bronchitis with scanty secretion.

3. R Sodii bicarb. gr. x.
Ammon. chlorid. gr. v.
Tr. hyoseyami ℥xv.
Sp. chloroformi ℥x.
Aquam ad ʒi.

In hot water, thrice daily.

4. R Potass. iodid. gr. iv.
Sp. ammon. arom. ℥xv.
Tr. camph. co. ℥xv.
Aq. chloroformi ad ʒi.

Thrice daily.

5. R Ammonii chloridi gr. v.
Ext. glycyrrhizæ fluidi ℥xv.
Aquam ad ʒii.

Every four hours for irritable cough.

6. R Terebene ℥x.-℥xv.
In capsules, or on sugar, thrice daily.

or R Olei terebinth. ℥xx.
Mucilag. acaciæ ℥ii.
Mist. amygdalæ ad ℥i.
Thrice daily.

7. R Tr. camph. co. ℥ss.
Oxymel scillæ ℥xv.
Mist. ammoniaci ad ℥i.
Thrice daily. In chronic
bronchitis of old people.

8. R Ammon. carb. gr. v.
Tr. nucis vom. ℥x.
Tr. cinchonæ co. ℥ss.
Sp. chloroformi ℥xv.
Aquam ad ℥i.
Thrice daily.

R Tr. ferri perchlor. ℥xv.
Liq. strychninæ ℥v.
Aq. chloroformi ℥i.
Thrice daily.
Useful tonics in later stages.

Catarrh (Common "Cold").

1. *Tr. aconiti* in ℥i. doses every
hour in early stages.

or R Potass. citrat. gr. x.
Sp. æth. nit. ℥xv.
Aquam ad ℥i.

Every four hours. Calomel
gr. i. at night, with a few grains
of Dover's powder, and saline
aperient on following morning.

2. Inhalation of vapour of equal
parts of oil of eucalyptus and
eau-de-Cologne in handker-
chief, or nasal spray of
glyco-thymoline (1 pt. to
3 of water).

3. R Tr. quininae ammon. ℥ss.
in water. A few doses
every four hours at outset.

4. R Morph. sulphatis gr. i.
Bismuth. subnit. ℥iii.
Pulv. acaciæ ℥i.

To be used as a snuff at the
beginning of a cold.

5. R Camphoræ ℥i.
Milk ℥ii.

Dissolve thoroughly. One tea-
spoonful in a little hot water
every three or four hours when a
cold threatens.

Chlorosis.

1. R Ferri sulph. exsic. gr. v.
Glycer. tragacanth. q.s.
Ft. pil. i.

Two to six or eight daily.

2. R Ferri sulph. gr. xxiv.
Magnes. sulph. ℥iv.
Acid. sulph. arom. ℥i.
Tr. zingib. ℥ii.
Aquam ad ℥viii.

Two tablespoonfuls twice a
day in chlorosis with constipa-
tion.

Chorea.

1. *Liq. arsenicalis* in gradually
increasing doses with or
without iron.

2. R Ammon. bromid. gr. x.-xx.
Chloral. hydrat. gr. x.-xx.
Aquam ad ℥i.

Occasionally as required.

or R Hyoscinae hydrobrom.
gr. $\frac{1}{10}$ - $\frac{1}{20}$.

Subcutaneously in severe
cases. Useful sedatives in
chorea.

Colic.

1. Hot fomentations, and warm
baths.

2. R Magnes. carb. ℥i.
Ammon. carb. ℥ss.
Tr. lavand. co. ℥ii.
Aq. menth. pip. ad ℥iv.

A tablespoonful occasionally
as required. In simple colic.

3. R Ol. ricini ℥vi.
Sodii bicarb. gr. xx.
Tr. opii ℥x.
Mucilaginis q.s.
Aq. cinnam. ad ℥ii.

A useful draught in colic

dependent upon irritating matter in bowel.

4. R Sod. sulpho-carbolat. gr. x.
Tr. belladonnæ.
Sp. chloroformi āā ℥ x.
Aquam ad ʒ i.

Thrice daily in flatulent colic.

5. R Magnes. carb. gr. i.
Syrup. zingib. ℥ x.
Sp. chlorof. ℥ i.
Aq. menth. pip. ad ʒ i.

Every hour, in colic of young children.

6. R Ext. opii gr. $\frac{1}{2}$ -i.
Ext. belladon. gr. $\frac{1}{4}$.
Ext. hyoscyami gr. ii.
Ft. pil. i.

Every four or six hours as required. In colic with intestinal obstruction.

7. *Enemata* of turpentine or castor oil are useful in colic with simple constipation.

Constipation.

1. R Ext. aloes socot. gr. i.
Ext. nucis vom. gr. $\frac{1}{2}$.
Ext. gentian. gr. iii.
Ft. pil. i.

A useful dinner pill in chronic constipation.

2. Decoction of aloes is a valuable aperient for females with pelvic troubles.

3. R Ext. belladon. gr. $\frac{1}{4}$.
Pil. aloes et myrrh. gr. iv.
Ft. pil. i.

Once or twice daily after meals in chronic constipation.

4. R Ext. cascara liq. ʒ i.
Tr. nucis vom. ʒ i.
Tr. belladon. ʒ ss.
Glycerini ad ʒ ii.

One teaspoonful, twice or three times a day in chronic constipation.

5. R Ext. aloes gr. ss.-i.
Ext. nucis vom. gr. ss.
Pulv. ipecac. gr. ss.
Pulv. capsici gr. i.
Ft. pil. i.

A useful dinner pill.

6. R Mist. sennæ co. ʒ i.-iss.

- or R Magnes. sulphat. ʒ ii.
Magnes. carb. gr. xx.
Aq. menth. pip. ad ʒ iss.

- or R Sodii phosphat. ʒ i.
Sodii bicarb. gr. xv.

- or R Seidlitz powder.

Taken in a tumbler of water in the morning.

Useful saline aperients.

7. R Pil. coloc. co.
Pil. hydrargyri āā gr. iiss.
Ft. pil. i.

- or R Pil. coloc. et hyoscyam.
gr. v. (B.P.).

- or R Pil. rhei co.
Pil. hydrargyri āā gr. iiss.
Ft. pil. i.

- or R Podophyllin gr. $\frac{1}{4}$.
Pulv. ipecac. gr. $\frac{1}{2}$.
Ext. gentianæ gr. iii.
Ft. pil. i.

- or R Podophyllin gr. $\frac{1}{4}$.
Pil. rhei co. gr. ii.
Ext. hyoscyam. gr. ii.
Ft. pil. i.

Useful aperient pills.

8. R Confect. sulphuris.
Confect. sennæ.

Confect. piperis æq. part.
A teaspoonful every morning.
Useful aperient in cases of piles.

9. R Pulv. jalapæ co. ʒ i.

Useful hydragogue purgative.
Effect increased by addition of calomel gr. i.

10. Pulv. scammon. co. (gr. v.-x.) with or without the addition of calomel, is a valuable aperient for dislodging intestinal worms.

11. Enemata—which may consist of simple soap and water, or contain turpentine or castor oil suspended in thin gruel.

Convulsions.

1. Warm baths.
2. Inhalation of chloroform.
3. R Potass. bromid. gr. v.
Chlor. hyd. gr. ii.
Aq. ad $\bar{z}$ i.

Add equal quantity of hot water, and inject per rectum. For convulsions of young children.

4. Morphia injected subcutaneously will often stop puerperal convulsions.

Cramp.

1. R Phenazoni gr. x.
At bed-time. For painful cramp in the legs.
2. R Tr. cannab. ind.
Ten drops on sugar at bed-time. For nocturnal cramp in gouty subjects.

Diabetes.

1. R Codeiæ gr. $\frac{1}{2}$.
or Ext. opii gr. $\frac{1}{2}$.
or Morphinæ acetat. gr. $\frac{1}{4}$.
In pill form, doses gradually increased, thrice daily.
2. R Sodii salicylat. gr. xx.
Liq. arsenical. $\mathbb{M}$ iii.
Aquam menth. pip. ad $\bar{z}$ i.
Thrice daily.
3. R Sodii phosphatis $\bar{z}$ i.
Sodii bicarb. gr. xv.
In a tumblerful of hot water every morning in some forms of gouty diabetes.
4. Washing with soap and hot water, followed up with use of borax lotion. For itching of skin about genitals.

Diarrhœa.

1. R Olei ricini $\mathbb{M}$ xxx. $\bar{z}$ i.
Sp. chloroformi $\bar{z}$ i.
Liq. morph. hyd. $\bar{z}$ i.
Pulv. acaciæ $\bar{z}$ i.
Aq. et syr. ad $\bar{z}$ iv.

One or two teaspoonfuls every one or two hours, as required. Valuable for general use.

2. R Bismuthi subnit. $\bar{\theta}$ i.
Pulv. ipecac. co. gr. v.
Thrice daily. In dysenteric diarrhœa of adults.

R Bismuthi subnit. gr. ii.
Pulv. ipecac. co. gr. $\frac{1}{2}$.
Pulv. cinnam. co. gr. ii.

Every three or four hours. In dysenteric diarrhœa of children.

3. Liq. arsenicalis in drop doses every three or four hours useful in lienteric diarrhœa.

4. R Tr. opii.
Tr. capsici $\bar{a}\bar{a}$ $\mathbb{M}$ i.
Sp. chloroformi $\mathbb{M}$ ii.
Aquam ad $\bar{z}$ i.

Occasionally as required. In diarrhœa persisting after expulsion of irritant.

5. R Acid. sulph. arom. $\mathbb{M}$ xv.
(Tr. opii $\mathbb{M}$ v.)
Aq. menth. pip. et syrup.
ad $\bar{z}$ i.

Every four hours. In summer diarrhœa of adults.

6. R Glycer. acid. carbol. $\mathbb{M}$ i.-ii.
Syr. rheados $\mathbb{M}$ xv.
Aquam ad $\bar{z}$ i.

Every two or three hours. In simple summer diarrhœa of children.

7. R Sodii sulphocarb.
Bismuthi subnit. $\bar{a}\bar{a}$ gr. ii.
Pulv. tragacanth. gr. $\frac{1}{2}$.
Glycerini $\mathbb{M}$ x.
Aq. anisi ad $\bar{z}$ i.

Every six hours, in infantile diarrhœa.

8. R Plumbi acetat. gr. ii.
Pulv. opii gr. $\frac{1}{4}$ - $\frac{1}{2}$.
Ext. hyoseyam. gr. ii.
Ft. pil. i.

As required. Valuable in obstinate cases.

9. R Liq. hyd. perchlor. ℥vi.
Aquam ad ʒi.

Every hour. In dysenteric diarrhœa with slimy bloody stools and tenesmus (children).

or Hydrarg. subchlor. gr. $\frac{1}{20}$.

Every three or four hours. In slimy offensive stools with vomiting (children).

10. *Opium* in various forms in dose according to age.
Dover's powder very useful.

11. R Zinci oxidi gr. ii.-iv.
(Tr. opii ℥ $\frac{1}{4}$ - $\frac{1}{2}$).
Aq. et mucilag. ad ʒi.

Every four hours in diarrhœa of children.

12. R Liq. ferri pernit. ℥xv.
Acid. nitric. dil. ℥xv.
Syrup. zingib. ʒss.
Aq. anethi ad ʒiss.

ʒi. every six hours.

Useful in chronic diarrhœa of children.

Dysentery.

1. Pulv. ipecac. gr. xx.-xxx. To be given in as small a quantity of fluid as possible. To be repeated in eight or ten hours if necessary. Give gr. x. at bed-time for two or three nights after.

2. R Magnes. sulphat. ʒi.
Acid. sulph. aromat. ℥x.
Aquam ad ʒi.

Every one or two hours, until motions become fœulent. Then

- R Acid. sulph. aromat. ℥x.
Tr. opii ℥x.
Sp. chloroformi ℥x.
Aquam ad ʒi.

Every four hours, till diarrhœa ceases.

3. R Liq. hydrarg. perch. ℥xii.
Aquam ad ʒii.

Every hour.

4. R Tr. benzoin. co. ʒss.
Tr. catechu ʒi.
Tr. opii ℥x.

Decoc. hæmatoxyli ad ʒi.

Thrice daily. Recommended in chronic dysentery.

5. R Liq. ferri pernitrat. ℥xv.
Sp. chloroformi ℥x.
Aquam ad ʒi.

Thrice daily in chronic dysentery.

6. R Argenti nitrat. gr. xx.-xl.
Aquæ destillat. Oi.

As an enema in chronic dysentery. To be followed by injection of morphia (gr. $\frac{1}{4}$).

Dyspepsia.

1. *Alkalis* with vegetable bitters very useful in various forms of dyspepsia. The addition of liq. arsenic. (℥i.-ii.) is useful in some forms of painful digestion.

- R Sodii bicarb. gr. xv.-xx.
Tr. hyoseyam. ℥xv.
Inf. calumb. *vel* quassia ʒi.

Useful in dyspeptic vertigo.

2. The *mineral acids* with vegetable bitters useful in atonic forms of dyspepsia.

3. R Sodii sulphitis ʒiss. *vel*
Sodii sulpho-carbol. ʒiss.
Tr. nucis vom. ʒi.
Aq. menth. pip. ad ʒviii.

Two tablespoonfuls thrice daily after food; in fermentative dyspepsia.

4. R Ammon. carb. gr. v.
Potass. bicarb. gr. x.
Inf. aurantii ad ʒi.

Thrice daily, for loss of appetite with flatulence.

5. *Liq. arsenicalis* in drop doses, useful in neuralgic gastrodynia. Also in morning vomiting of drunkards.

6. *Bismuth* mixtures are useful in painful digestion. The addition of hydrocyanic acid is often desirable.

7. R Pulv. rhei $\bar{3}$ ss.
Sodii bicarb. $\bar{3}$ iss.
Aquæ menth. pip. $\bar{3}$ iv.

$\bar{3}$ ss before meals in dyspepsia with constipation.

8. R Ext. aloes socot. gr. $\frac{1}{2}$.
Ext. gentian. gr. i.
Ext. belladon. gr. $\frac{1}{4}$.
Ext. nucis vom. gr. $\frac{1}{2}$.
Ft. pil. i.

A useful dinner pill.

9. R Ext. nucis vom. gr. $\frac{1}{2}$.
Pulv. ipecac. gr. $\frac{1}{2}$.
Ext. gentian. gr. ii.
Ft. pil. i.

An excellent dinner pill in slow digestion.

10. R Bismuthi subnit.
Carbonis ligni $\bar{a}\bar{a}$ gr. v.
In extreme flatulence. Three or four times daily.

Emphysema.

1. R Tr. ferri acetat. $\mathbb{M}$ xv.
Sp. chloroformi $\mathbb{M}$ xx.
Aquam ad $\bar{3}$ i.
Thrice daily.

2. R Potass. iodid. gr. v.
Ammon. carb. gr. v.
Infusi gentian. co. $\bar{3}$ i.
Thrice daily.

3. Cod-liver oil.

4. Arsenic—systematic course.

Epilepsy.

1. R Potass. bromid. gr. x.-xx.
Liq. arsenicalis $\mathbb{M}$ ii.
Aq. ad $\bar{3}$ i.
Thrice daily, after meals.

2. R Potass. bromid. gr. xv.
Tr. belladonnæ $\mathbb{M}$ v.-x.
Aquam ad $\bar{3}$ i.

Thrice daily when bromides fail, taken alone.

3. R Liq. trinitrin. $\mathbb{M}$ i.
Aquam ad $\bar{3}$ i.

Three or four times a day for minor epilepsy. Inhalation of nitrite of amyl sometimes useful for *status epilepticus*.

4. R Zinci oxidi gr. ii.-iii.
Ext. glycyrrhizæ q.s.
Ft. pil. i.

One or two thrice daily.

5. R Zinci valerianat. gr. i.-ii.
Ext. belladon. gr. $\frac{1}{4}$.
Ext. glycyrrhizæ q.s.
Ft. pil. i.

Thrice daily.

6. R Boracis gr. x.
Glycerini $\bar{3}$ i.
Aquæ $\bar{3}$ i.

Thrice daily. In chronic cases when bromides fail.

Erysipelas.

Useful external applications :—

1. Ung. ichthyol 25-50 per cent.

2. R Acid. carbol. $\bar{3}$ ss.
Sp. vini rect. $\bar{3}$ ss.
Tr. iodi $\bar{3}$ i.
Glycerini $\bar{3}$ x.

Paint on skin occasionally.

3. Collodion flexile.
Excellent for ordinary case.

4. R Zinci oxidi
or Calamini $\bar{3}$ ii.
Pulv. tragac. $\bar{3}$ ii.
Acid. boric. $\bar{3}$ i.
Glycerini $\bar{3}$ i.

Aq. bullientis ad $\bar{3}$ vi.
To be brushed (when cold) over the affected part, and allowed to dry.

Fever.

1. R Sp. æth. nit. ℥xx.
Liq. ammon. acet. ℥i.
Aquam ad ℥i.
Every four or six hours.
- or R Potass. nitrat. gr. v.
Vin. antimon. ℥v.
Liq. ammon. acetat. ℥ii.
Aq. camph. ad ℥i.
Every four hours.
- or Solution of bitartrate of potash (a teaspoonful to the pint), sweetened and flavoured with lemon juice.
Useful salines in mild fever.
2. R Potass. chloratis ℥i.
Acid. hydrochlor. dil. ℥i.
Syrup. zingiber. ℥i.
Decoc. hordei Oii.
- or R Acid. phosphor. dil. ℥iii.
Glycerin. ℥i.
Decoc. hordei Oii.
Useful acid drinks in mild fevers.
3. *Tr. Aconiti*—in drop doses every half-hour, or hour, very useful in early stages of fever.
4. *Alcohol*—in later stages of fever, with nervous depression, muttering delirium, and compressible pulse.
5. *Quinine*—gr. x.-xx., *antipyrin*—gr. xx.-xxx., or *antifebrin*—gr. ii.-v. Best given about bed-time. As antipyretics.
6. *Cold baths*, wet-sheet packs, or sponging with tepid or cold water; very useful in reducing temperature.

Gall-stones.**DURING THE PAROXYSM.**

1. *Opium*, or preferably subcutaneous injections of morphia.

2. R Tr. belladonnæ ℥x.
Aquæ menth. pip. ℥ss.
Every hour, till well-marked physiological effects are obtained.

3. Inhalation of chloroform.**DURING THE INTERVAL.**

4. R Ammon. chlorid. gr. x.
Succi taraxaci ℥i.-ii.
Aquam ad ℥i.
Thrice daily.
5. R Sodii phosphat. ℥i.
Aquæ ℥i.
Thrice daily.

Gastralgia.

1. R Acid. arseniosi gr. $\frac{1}{15}$.
Ext. gentian. q.s.
Ft. pil. i.
Thrice daily.
2. R Bismuthi carb. gr. x.
Sodii bicarb. gr. x.
Pulv. tragac. co. gr. v.
Acid. hydrocyan. dil. ℥iii.
Aq. menth. pip. ℥i.
Thrice daily, before meals.
3. *Charcoal*—in dose of gr. xxx. in gastrodynia with much flatulence.
4. *Pepsin*—in dose of gr. v.-x., with meals, in gastrodynia, following the use of animal food.

Gout.

1. Calomel at bed-time, followed by a saline aperient draught in the morning, necessary at the outset.
2. R Potassii citrat. gr. xx.
Vini colchici ℥xx.
Aq. menth. pip. ad ℥i.
Every four hours. Omit colchicum as soon as pain is relieved.

3. R Sodii salicylatis gr. xx.
Vini colchici ℥ xv.
Aquæ menth. ℥ i.

Every four or six hours in acute gout.

4. R Magnesii sulph. ℥ ss.
Magnesii carb. gr. x.
Vini colchici ℥ xv.
Aquæ menth. pip. ℥ i.

Every four or six hours in acute gout, with constipation.

5. R Potassii bromid. gr. xx.
Tr. cannab. ind. ℥ xv.
Aq. camph. ad ℥ i.

At bed-time, for sleeplessness. Opium contra-indicated.

6. R Sodii bicarb. ℥ ss.
Tr. opii ℥ ss.
Aquam ad Oss. Ft. lotio.

Mix with equal part of hot water. As local application to inflamed joints.

7. R Potass. iodid. gr. v.-x.
Sp. ammon. arom. ℥ xv.
Vini colchici ℥ v.-x.
Infusi cinchonæ ℥ i.

Thrice daily. In chronic gout.

8. *Piperazin*—Five grains in a glass of aerated water three times a day has been recommended in chronic gout.

9. *Mineral waters* of Vichy, Wiesbaden, and Carlsbad.

Graves' Disease.

1. R Tr. belladonnæ ℥ x.
Tr. strophanthi ℥ viii.
Aquam ad ℥ i.

Thrice daily.

2. Calcium chloride, in gr. x. doses, thrice daily, is sometimes of service.

Hæmorrhage.

1. *Cold*—as an external application, or by sucking ice.

2. R Acid. gallic. gr. x.-xv.
Acid. sulph. arom. ℥ xv.
Tr. cinnamon. ℥ ii.
Aquam ad ℥ i.

As a draught, every four or six hours.

- R Calcii chloridi gr. x.
Aq. ad ℥ i.

Thrice daily. In hæmoptysis.

3. R Ext. ergot. liq. ℥ ss.-℥ i.
Aquam ad ℥ iss.

As a draught occasionally as required.

4. R Ferri ammon. sulph. gr. v.
Aquæ ℥ i.

Every six or eight hours. An excellent styptic in hæmatemesis.

5. R Ol. terebinth. ℥ x.-xx.
Mist. amygdalæ ℥ i.

Every three or four hours. In hæmaturia smaller doses to be used (℥ iii.-v.) and with caution.

6. R Infus. maticæ ℥ iss.

Three or four times daily in hæmaturia.

7. *Tr. hamamelidis*—℥ ii.-iv. in water every two or three hours serviceable in hæmoptysis and hæmatemesis.

Headache.

BILIOUS HEADACHES.

1. R Quin. sulph. gr. ii.
Pulv. rhei gr. iii.
Mucilaginis q.s.
Ft. pil. i.

To be taken at bed-time.

2. R Sodii bicarb. gr. xv.
Tr. nucis vom. ℥ vi.
Inf. gentian. co. ℥ i.

Thrice daily before meals.

3. R Infusi sennæ.
Infusi gentian co. āā ℥ ss.

Once or twice daily.

4. R Pil. hydrarg.
Pil. rhei co. āā gr. iv.
Ext. hyoscyam. gr. ii.
Ft. pil. ii.

At bed-time.

CONGESTIVE HEADACHES.

1. Saline aperients.
2. R Ferri sulph. gr. ii.
Magnesii sulph. gr. xxx.
Acid. sulph. dil. ℥xii.
Tr. card. co. ℥i.
Aquam ad ℥i.
Thrice daily.
3. R Tr. ferri perchlor. ℥xv.
Acid. hydrochlor. dil. ℥x.
Sp. chloroform. ℥x.
Thrice daily, especially during climacteric period.
4. R Tr. veratri virid. ℥i.
In water every hour. Useful in congestive headaches recurring at menstrual period.

NERVOUS AND SICK HEADACHES.

1. R Potass. bromid. gr. x.
Phenazoni gr. v.-x.
Aquæ camph. ℥i.
Thrice daily in nervous headaches with wakefulness.
2. R Ext. cannab. ind. gr. $\frac{1}{4}$ - $\frac{1}{2}$.
(Camphor. gr. i.).
Confect. rosæ q.s.
Ft. pil. i.
At bed-time, or twice daily. In nervous headaches, with troubled sleep.
3. R *Tr. nucis vom.*—in dose of ℥i. every half-hour or hour in headache from exhaustion.
4. *Strong tea or coffee* in small quantity for nervous exhaustion. Contra-indicated if it causes palpitation.
5. *Guarana*—℥ss.-i. of the powder, in typical migraine.
6. R Zinci oxidi gr. i.-ii.
Confect. rosæ q.s.
Ft. pil. i.
Thrice daily after meals. In nervous headaches.

7. *Phosphorus*—gr. $\frac{1}{50}$ - $\frac{1}{30}$ in the form of a pill, thrice daily. In nervous headache.

NEURALGIC HEADACHES.

1. R Quininæ sulph.
Camphoræ
Ext. hyoscyami āā gr. i.
Confect. rosæ q.s.
Ft. pil. i.
A very useful pill; to be taken thrice daily.
2. R Butyl-chloral. hyd. gr. v.
Three or four times daily.
3. R Liq. arsenici hyd. ℥v.
Tr. ferri perch. ℥v.
Aquam ad ℥i.
Thrice daily after meals.
4. R Ung. aconitinæ.
or R Ung. veratrinæ.
Useful local applications.

ORGANIC HEADACHES.

1. R Potass. iodid. gr. v.-xv.
Potass. brom. gr. x.
Inf. calumb. ℥i.
Thrice daily, in syphilitic headache.
2. Bromide of potassium (*vide supra*).
3. Opium and morphia, carefully used, sometimes give great relief.

Heart Disease.

1. Digitalis prescriptions. The tincture is the best preparation, as a rule.
R Tr. digitalis ℥x.
Sp. æth. nit. ℥xx.
Aquam ad ℥i. Thrice daily.
- R Tr. digitalis ℥x.
Tr. nucis vom. ℥vi.
Sodii nitritis gr. ii.
Aquam ad ℥i. Thrice daily.

- R Pulv. digitalis gr. $\frac{1}{2}$ -i.
 Pulv. scillæ gr. i.
 Pil. hydrarg. gr. i.
 Conf. rosæ q.s. ft. pil. i.
 Thrice daily.

Digitalis to be suspended if it causes or increases irregularity of pulse.

2. Tr. strophanthi (℥v.-x.) preferable to digitalis with high arterial tension or cardiac degeneration, and in aortic disease. Strophanthin (gr. $\frac{1}{30}$ - $\frac{1}{15}$), hypodermically injected, indicated when rapid effect is desired.

3. For severe cardiac dyspnoea:—*Morphia*—in small doses. Should be used cautiously, and not frequently repeated.

Nitrites—inhalation of nitrite of amyl; drop doses of liq. trinitrin. : or sodium nitrite.

Strychnia—hypodermic injection of gr. $\frac{1}{30}$.

4. Calomel, together with digitalis in some form, is indicated in cardiac dilatation with dropsy.

5. *Strychnia*—Useful cardiac tonic. In syncopal cases, ℥i. every hour for ten doses then less frequently. Subcutaneously gr. $\frac{1}{30}$ - $\frac{1}{6}$ in threatening cardiac failure (usefully combined with inhalation of oxygen).

6. Paraldehyde ℥i.
 or Chloralamide ℥ss.
 or Chloralose gr. v. (in cachet)
 or Veronal gr. v. (in cachet)
 or Trional gr. x.-xx.

Useful hypnotics in chronic valvular disease.

7. Diuretin gr. xv.
 Thrice daily.
 or Caffein citrate gr. ii.-iv.

Useful diuretics in valvular disease.

8. *Liq. arsenicalis*—℥iii.-v., well diluted, thrice daily, for cardiac pain and distress.

Hiccough.

1. Sucking *ice*.
2. *Morphia*—hypodermically injected.
3. R Chloral hydrat.
 Potass. bromid.
 Potass. bicarb. āā ℥i.
 Aq. menth. pip. ad ℥vi.
 One tablespoonful every four hours. Useful in some obstinate cases.
4. *Bismuth*—with alkalies and dilute hydrocyanic acid, before meals, associated with acidity.
5. *Phenazone*—gr. v. in water, every four or six hours.
6. *Gastric lavage*—in some obstinate cases.

Hysteria.

1. R Potass. bromid. gr. xv.-xx.
 Tr. lavandulæ co. ℥xx.
 Sp. ammon. arom. ℥xx.
 Inf. valerian. ad ℥i.
 Thrice daily, as a general calmative.

2. *Phosphorus*—in dose of gr. $\frac{1}{30}$ to $\frac{1}{10}$, with or without the addition of strychnia or nuxvomica, is an excellent tonic in hysteria.

3. R Liq. strychninæ ℥iv., gradually increased.
 Tr. card. co. ℥xv.
 Aquæ camph. ℥i.

Thrice daily, in hysterical paralysis. Paralysed muscles to be faradised.

4. R Zinci phosphid. gr. $\frac{1}{6}$ - $\frac{1}{3}$.
Ext. nucis vom. gr. $\frac{1}{2}$.
Pil. aloes cum myrrh.
gr. iii.

Twice or thrice daily, in hysteria with constipation.

Jaundice.

1. A mercurial aperient pill at night, followed by a saline aperient draught in the morning, is beneficial in catarrhal jaundice.
2. Benzoate of soda, phosphate of soda, or ammonium chloride, may be given in catarrhal jaundice.
3. R Bismuthi carb. gr. x.
Sodæ salicylati gr. x.
Tr. rhei $\overline{3}$ ss.
Aquam menth. pip. ad $\overline{3}$ i.
Thrice daily, in catarrhal jaundice.
4. R Hydrarg. $\overline{c}$. creta gr. $\frac{1}{6}$ - $\frac{1}{3}$.
Sacchari alb. q.s.
Thrice daily, together with
R Acid. nitro-mur. dil. $\mathbb{M}$ vi.
Tr. nucis vom. $\mathbb{M}$ vi.
Aquam ad $\overline{3}$ i.
Useful in chronic catarrhal jaundice.
5. Sulpho-carbolate of soda, ten to twenty grains, thrice daily in half a tumblerful of hot water.
6. Hot air or water bath, or hot wet-sheet pack; followed by sponging with a weak alkaline lotion. To relieve itching of the skin.

Laryngismus Stridulus.

1. Warm baths—during paroxysm.
2. Chloroform—inhalation during paroxysm, if very severe.

3. Potassium bromide, with or without the addition of chloral, may be given by mouth or rectum with advantage.

4. Cod-liver oil and other tonics are useful in chronic cases.

Laryngitis.

1. Hot fomentations to throat externally, with inhalations of steam, in acute forms.
2. R Liq. ammon. acetat. $\overline{3}$ i.
Liq. morph. hydroch.
Vin. antimon. $\overline{a}\overline{a}$ $\mathbb{M}$ vi.
Succi conii $\mathbb{M}$ xx.
Aquam camph. ad $\overline{3}$ i.
Every four hours. In acute laryngitis of adults.
3. R Tr. benzoin. co. $\overline{3}$ i.
Chloroform, a few drops.
Added to boiling water. For inhalation in acute laryngitis.
4. A spray of equal parts of 1 per cent. sol. of cocaine, and 1 per cent. sol. of carbolic acid, is useful to allay irritation of the throat.
5. Useful astringent spray-solutions in chronic laryngitis :
Alum (gr. v. ad $\overline{3}$ i.).
Tannin (gr. x. ad $\overline{3}$ i.).
Tr. ferri perchlor. ($\mathbb{M}$ x. ad $\overline{3}$ i.).
Sulphate of zinc (gr. ii. ad $\overline{3}$ i.).
6. Soothing inhalations in chronic laryngitis :—
Ammonium chloride vapour.
Vapor conii (B.P.).
Eucalyptol.
Carbolic acid.
Terebene.

Lead-poisoning.

1. Hot fomentations to abdomen in lead colic.

2. *Aperients*—of which magnesium sulphate is the best for continuous use, and castor oil for temporary use.

3. *Morphia*—gr. $\frac{1}{6}$ to $\frac{1}{3}$ hypodermically injected, with or without liq. atropinæ sulph. $\mathbb{M}$ i.-ii. in lead colic.

4. R Potass. iodid. gr. v.-x.
Inf. calumbæ $\mathfrak{z}$ i.
Thrice daily in chronic cases.

5. R Sodii chlorid. $\mathfrak{z}$ ss.
Thrice daily, dissolved in water, has also been recommended in chronic cases.

Liver—Diseases of.

1. *Aperients*—Salines are the best, as magnes. sulph., sodæ sulph., sod. pot. tart., and purgative mineral waters. An occasional mercurial pill is sometimes of great benefit. Podophyllin not so generally useful.

2. *Alkalies*—in the form of carbonates, or bicarbonates, in combination with vegetable bitters.

3. *Chloride of ammonium*—in doses of not less than gr. x. thrice daily, is of high value. It may be combined with acids or alkalies.

4. *Mineral acids*—especially the nitro-muriatic in small doses ($\mathbb{M}$ v.) thrice daily; often of great service.

Locomotor Ataxy (*Tabes dorsalis*).

1. R Phenazon. gr. v.-x.
Aquæ $\mathfrak{z}$ i.

Thrice daily.

or R Ext. belladon. gr. $\frac{1}{4}$.
Ext. cannab. ind. gr. $\frac{1}{4}$.
Camphoræ gr. i.
Confect. rosæ q.s.
Ft. pil. i.

Occasionally as required. To relieve the pains of early stages.

2. R Liq. auri et arsen. brom.
or Liq. auri et hydrarg. brom.

Ten drops in water, thrice daily.

Lumbago.

1. R Sodæ salicylat. gr. xx.
Aq. menth. pip. ad $\mathfrak{z}$ i.

Single dose will sometimes give great relief if given soon after onset.

2. R Sodii salicylat. gr. x.
Potass. citrat. gr. xx.
Aq. menth. pip. ad $\mathfrak{z}$ i.

Three or four times a day.

3. A pad of cotton-wool, well sprinkled with sulphur, applied to the loins, after hot poultice or fomentations.

4. Hot baths.

5. Stimulating liniments, with thorough friction.

6. R Phenazoni gr. x.
Chlorodyne $\mathbb{M}$ x.
Aquæ camphoræ $\mathfrak{z}$ i.

Useful sedative draught at night, in acute cases.

7. Constant electric current daily.
Useful in chronic cases.

Lymphatic Glands—chronic enlargement of.

1. R Liq. arsenicalis $\mathbb{M}$ iii.
Potassii iodidi gr. iii.
Aquam ad $\mathfrak{z}$ i.

Thrice daily in water after food. Increase dose gradually.

2. *Sodium cacodylate*—A carefully prepared hypodermic solution containing .05 grm. cacodylic acid in 1 c.c., injected once daily, is sometimes worth trial.

Measles.

1. *Aconite*—in drop doses of the tincture every hour. Very useful at the onset.
2. R Vin. ipecac. ℥ss.
Tr. camph. co. ℥iss.
Liq. ammon. acet. ℥ss.
Syrupi ℥ss.
Aquam ad ℥ii.

A teaspoonful every two hours. Useful in the catarrhal stage (children).

3. R Liq. ammon. acet. ℥xv.
Sp. ætheris nit. ℥x.
Aquam ad ℥ss.

Every four or six hours. A mild diaphoretic in simple cases.

4. R Potass. citrat. gr. v.-x.
Vini ipecac. ℥v.-x.
Tr. camph. co. ℥v.-x.
Aquam ad ℥ii.

A simple cough mixture for early stages.

5. R Potass. chlorat. gr. v.
Acid. hydrochlor. dil. ℥iii.
Syrupi limonis ℥ss.
Aquam ad ℥ss.

Every three or four hours. In measles of low type, with brown dry tongue and prostration (children).

6. *Bromide of potassium*—useful as a sedative, and to procure sleep in cases with active nervous symptoms and wakefulness.

7. *Tepid bath* or *tepid wet sheet-pack* useful in high fever, and for early delirium.

8. *Mustard baths*—useful on sudden retrocession of the rash.

Meningitis.

1. *Active purge*—desirable at outset of the acute form, *e.g.* :—

A grain of calomel, followed in two hours by saline aperient.

2. Head to be shaved, and ice applied, or leeches behind ears in acute cases.

3. R Potass. bromid. gr. v.
Phenazon. gr. ii.-v.
Aq. ad ℥ii.

For relief of headache. To be repeated as required. In tubercular meningitis.

4. R Potass. iodidi gr. ii.-v.
Ammon. bromid. gr. ii.-v.
Syrupi aurantii ℥i.
Aq. ad ℥ii.

Every four or six hours.

5. *Mercurial inunctions* — in syphilitic cases.

Migraine.

1. Phenazon. gr. v.-x.
or Phenacetine gr. v.-x.

To relieve pain. Repeat as required.

2. *Sodium salicylate*—in doses of gr. xx. given in half a tumblerful of water with a dessertspoonful of effervescing citrate of caffeine will sometimes cut short an attack.

3. Pil. hydrarg. gr. v. overnight and saline aperient following morning when attack threatens.

4. R Acid nitro-mur. dil. ℥i.
Add to a tumblerful of water. Half to be taken at once, and remainder in half an hour (Haig). To relieve an attack.

Myalgia.

1. *Lin. belladon.* or *lin. opii.*
Useful external applications.

2. R Ammon. chlorid. gr. x.-xx.
Aqua ℥i.

Thrice daily. Highly recommended by Dr. Anstie.

3. R Potass. nitratis gr. v.-x.
Pulv. ipecac. co. gr. iii.
To be taken as a powder every
four hours.

Neuralgia.

1. R Ung. veratrinæ.
R Ung. aconitinæ.
R Morphinæ gr. i.-ii.
Acid. oleic. ℥i .
R Lin. opii.
R Lin. aconiti.
R Lin. belladon.
Useful local applications in
neuralgia.
2. *Hypodermic injections* of mor-
phia or atropia, or of both
combined.
3. R Quin. sulph. gr. i.
Ext. hyoseyam. gr. i.
Camphoræ gr. i.
Confect. rosæ q.s.
Ft. pil. i.
Every four hours.
4. R Liq. arsenici hyd. ℥v .
Tr. ferri perchlor. ℥v .
Sp. chloroformi ℥x .
Aquam ad ℥i .
Thrice daily after meals.
5. R Tr. gelsemii ℥xv.-xx .
Aquam ad ℥ss .
Every four hours in toothache.
6. R Butyl-chloral hydr. gr. v.
Syrupi aurantii ℥ss .
Aquam ad ℥ss .
Occasionally as required.
7. *Quinine, antipyrin or phenace-
tin*—in a single dose, during
a paroxysm.
8. *Phosphorus*—in doses of gr.
 $\frac{1}{100}$ to $\frac{1}{30}$ every three hours.
Useful in many forms of
neuralgia.
9. *Faradisation* of painful spot.

Otitis.

1. Careful cleaning, followed by

insufflation of boracic acid
or iodoform. Naso-pharynx
to be carefully examined,
and dealt with as required.

2. *Liq. hydrarg. perchlor.* B.P.
Useful local application in
inflammation of external
meatus caused by small
boils:

3. R Lot. plumbi sub-acet. dil.
or R Lotio zinci sulph. (gr. ii.
ad ℥i .)

or R Boroglyceride— ℥i . to a
wineglassful of warm
water.

Useful lotions for the ear
passage in otorrhœa.

4. A small *blister behind the ear*
is often beneficial in severe
otitis.

5. *Tonics and Cod-liver oil* often
of service in chronic cases.

Ozæna.

1. Thorough removal of crusts
by irrigation with warm
water solution of bicarbon-
ate of soda and borax (gr. v.
of each to ℥i .) followed by :
Carbolic acid (1 per cent.
solution).

or Formalin (℥v.-x . ad aq. Oss.)
or Boroglyceride (℥ss . ad aq. Oi.)
or Sanitas (℥i . ad aq. Oi.).

2. *Ichthyol*—(25 to 50 per cent.
in paraffin liq.), used as a
spray.

3. *Tonics and Cod-liver oil* are
often required.

Paralysis.

1. *Liq. strychninæ*—in doses of
 ℥iii . internally, thrice daily
or of ℥i . hypodermically,
once daily, the quantity
being gradually increased
—is more generally useful
than any other drug.

2. R Potass. iodid. gr. v.-xx.
Ammon. carb. gr. ii.
Infusi calumbæ $\bar{z}$ i.

Thrice daily. Useful in syphilitic and some chronic forms of paralysis.

3. *Phosphorus*—in dose of gr. $\frac{1}{100}$ to $\frac{1}{30}$ in pill, twice or thrice daily; recommended in hysterical paralysis.

- or R Zinci phosphid. gr. $\frac{1}{3}$.
Ext. nucis vom. gr. $\frac{1}{2}$.
Ext. gentian. gr. ii.
Ft. pil. i.

A useful pill in similar cases.

4. R Potass. bromid. gr. x.-xx.
Tr. belladon. $\mathbb{M}$ x.
Aquam camph. ad $\bar{z}$ i.

Thrice daily. In paralysis accompanying acute congestion of nerve-centres.

5. *Galvanism* or *faradism* of affected muscles useful in chronic stage of peripheral paralysis. That form of application should be selected to which the muscles best respond. No use where the irritability has quite disappeared.

6. *Counter-irritation* of the spine exceedingly useful in diphtheritic paralysis and other chronic forms.

Perspiration, profuse.

1. *Liq. atropinæ sulph.*—in dose of $\mathbb{M}$ $\frac{1}{2}$ -i. hypodermically injected is the promptest remedy.

- or R Ext. belladon. gr. $\frac{1}{2}$.
Ext. gentian. gr. ii.

To be taken at bed-time.

2. R Zinci oxid. gr. ii.
Ext. belladon. gr. $\frac{1}{4}$.
Glycerini tragac. q.s.

For a pill to be taken at bed-time.

3. R Acid. camphoric. $\bar{z}$ ss.
Suspended in warm milk.
Occasionally at bed-time.

4. R Pulv. ipecac. co. gr. v.
As a powder, the last thing at night. If made as a pill, ext. belladon. gr. $\frac{1}{4}$ may be added with advantage.

5. A level teaspoonful of precipitated sulphur in milk, twice daily. Good for excessive general sweating. If it purges, add pulv. cretæ aromat.

Pharyngitis.

ACUTE (*Sore-throat*).

1. After a saline aperient or a grain of calomel.

- R Sodæ salicylatis gr. xx.
Sp. æth. nitrosi $\bar{z}$ i.
(Liq. morph. hydrochl. $\mathbb{M}$ x.)
Aquam ad $\bar{z}$ i.

At bed-time.

2. R Acid. carbol. $\bar{z}$ i.
Cocain. hydrochlor. gr. xx.
Glycerini boracis $\bar{z}$ i.
Aq. ad $\bar{z}$ xii.

A useful gargle.

3. Cleansing the throat with solution of borax and subsequent application of cocaine (5 per cent. solution) spray. When much pain.

4. Astringent sprays (*v.* Laryngitis).

CHRONIC.

1. Avoid strong alcoholic drinks and smoking of tobacco.

2. Solution of silver nitrate (gr. iii.-x. to the ounce); chloride of zinc (gr. v.-x. to the ounce); and glycerine of tannin, are useful local applications on occasion.

3. R Glycer. acid. carbolic. $\bar{z}$ iii.
Acid. tannic. $\bar{z}$ ii.
Tr. capsici $\bar{z}$ i.
Infusi rosæ acid. ad $\bar{z}$ xii.

A useful gargle. Dilute if necessary.

4. Electro- or thermo-cauterisation of enlarged follicles, most useful in very chronic cases.

Phthisis.

1. *Creosote*—in capsule, thrice daily. Begin with not more than $\mathbb{M}$ i. for a dose, and gradually increase.

2. R Quininæ sulph. gr. i.
Pulv. digitalis gr. i.
Pulv. opii gr. $\frac{1}{2}$.
Ft. pulv.

Every four or six hours, in acute phthisis. To be accompanied with the application of iced cloths to the abdomen. (Anderson.)

3. R Sodii hypophosphit. gr. vi.
Syrupi $\bar{z}$ ii.
Aquam ad $\bar{z}$ i.

- or R Calcii hypophosphit. gr. vi.
Syrupi $\bar{z}$ ii.
Aquam ad $\bar{z}$ i.

- or R Sodii hypophosph.
Calcii hypophosph. $\bar{a}\bar{a}$ gr. vi.

Infusi cascarillæ $\bar{z}$ i.

Twice a day after meals.

Of no use in acute or very advanced phthisis. These medicines should be kept in a dark place.

4. *Cod-liver oil*—in doses of $\bar{z}$ i.-iv. thrice daily. The addition of ether to the oil ($\bar{z}$ iiss. ad $\bar{z}$ iv.) has been recommended.

5. *Guaiacol carbonate*—in doses of gr. v.-x. in milk, thrice

daily, after meals. Sometimes of great service in pulmonary phthisis.

6. *Pancreatic emulsion* (Dobell). A tablespoonful from one to two hours after meals in milk.

7. R Picis liquid. gr. ii.
Pulv. glycyrrhiz. co. q.s.
Ft. pil. i. Three or four times daily for profuse fœtid expectoration.

8. R Sodii bicarbonat. gr. xv.
Aq. menth. pip. $\bar{z}$ i.
To be sipped occasionally—
for irritable cough.

or *Codeinæ* gr. i.—dissolved in an ounce of calf's foot jelly. A teaspoonful occasionally for irritable cough.

- or R Heroin hydroch. gr. $\frac{1}{2}$ - $\frac{1}{6}$.
Syr. pruni virgin. $\bar{z}$ ss.
Aq. menth. pip. ad $\bar{z}$ ii.
Ft. linct.

Occasionally at night, when sleep is prevented by cough.

9. *Counter-irritation*—by iodine painted under clavicles; in chronic forms: to allay cough and to check secretion.

10. For night-sweats, see "*profuse perspiration*."

11. For hæmoptysis, see "*hæmorrhage*."

Pleurisy.

1. R Liq. ammon. acet. $\bar{z}$ i.
Potassii citrat. gr. xv.
Sp. æth. nit. $\mathbb{M}$ xx.
Aq. ad $\bar{z}$ i.

Every four hours. A useful saline in early stages.

2. R Sodii salicylat. gr. x.-xv.
Sodii citrat. gr. x.-xv.
Aq. menth. pip. ad $\bar{z}$ i.

Every four hours.

3. To relieve pain in side :—

Hot fomentations.

Application of *lecches*.

Hypodermic injection of *morphia* (gr. $\frac{1}{4}$).

R Phenazon, gr. v.
Potassii brom. gr. x.
Tr. opii ℥v.
Aq. ad ʒss.

Every three or four hours, for severe pain and restlessness in early stage.

4. *Croton oil liniment*.—A useful external application to promote absorption of fluid.

5. *Strapping* the side with strips of soap plaster greatly relieves late pains, after absorption of fluid.

6. *Tonics*—especially the syrup of the iodide of iron and cod-liver oil.

In convalescence.

Pneumonia.

1. *Tr. aconite*—one drop every half-hour for four hours, then less frequently. For children.

Quinine—in single dose of gr. v.-x. together with gr. $\frac{1}{4}$ to $\frac{1}{3}$ of *morphia*, followed every three or four hours by smaller doses without *morphia*.

Recommended at first onset of acute pneumonia.

2. R Liq. ammon. acet. ʒss.-i.
Vin. ipecac. *vel*.
Vin. antim. ℥x.-xv.
Liq. morph. ℥x.-xv.
Aquam ad ʒi.

Every four or six hours. A useful formula during early acute stages, with much pain and restlessness.

3. R Ammon. carb. gr. v.
Sp. chloroform. ℥xx.
Inf. cinchonæ ad ʒi.

Every three or four hours. In acute pneumonia of the aged and feeble.

4. Hot fomentations and turpentine stupes to relieve pain of side.

5. R Ammon. carb. gr. $\frac{1}{2}$ -i.
Vin. ipecac. ℥ii.
Tr. aconiti ℥i.
Aq. et syrup. ad ʒi.

Every three or four hours. Useful in acute pneumonia of young children (early stages).

6. Hot mustard bath invaluable in many cases of acute pneumonia and bronchitis in children.

7. R Tr. digitalis ℥viii.
Tr. belladon. ℥iv.
Tr. nucis vom. ℥vi.
Aquam ad ʒi.

Every six or eight hours, if cardiac failure threatens.

8. For sleeplessness :—

Hypodermic injection of morphia. For adults only. In early stages.

R Trional gr. xx. (or more).

Dissolved in hot whisky, and then diluted with water.

R Chloralamide gr. xxx.-xv.

9. For delirium :—

Graduated cold bath or *cold wet sheet-packs*—in young adults with high temperature.

A liberal allowance of alcohol in warm milk—in weakly, alcoholic, or aged patients.

10. Hypodermic injection of liq. strychninæ (℥iii.-v.), and oxygen inhalations are useful when cardiac failure threatens.

Rheumatism, acute.

1. R Salicin. gr. xx.
Aquaë $\bar{z}$ i.
Every two or three hours.
2. R Sodii salicylat. gr. x.-xx.
Sodii bicarb. gr. x.-xx.
Aquaë $\bar{z}$ i.
Every two or three hours.
3. R Sodii bicarb. $\bar{z}$ i.
Potass. acetat. $\bar{z}$ ss.
Liq. ammon. acetat. $\bar{z}$ i.
Aquaë $\bar{z}$ ii.
Every four, six, or eight hours
in effervescence with acid. citric,
 $\bar{z}$ ss. till the urine is alkaline;
then less frequently. (Fuller.)
4. Acetanilide gr. iii.-vi.
Thrice daily. For relief of
pain, when for any reason
salicyl drugs are not available.
5. R Mist. quininæ $\bar{z}$ i.
Thrice daily during convales-
cence.
6. R Ext. opii gr. i.-ii.
Ext. belladon. gr. $\frac{1}{4}$ - $\frac{1}{2}$.
Ft. pil. i.
At bed-time if great restless-
ness and pain.
7. R Guaiacol.
Glycerini æq. partes. M.
Smear over joint, cover with
lint, and bandage.
3. *Tonics*—during convalescence.
R Ammon. carb. gr. v.
Tr. lavand. co. $\bar{z}$ i.
Infusi cinchon. $\bar{z}$ i.
Thrice daily. An excellent
formula.

Rheumatism, chronic.

1. R Potass. iodid. gr. v.-x.
Ammon. carb. gr. ii.
Inf. cahumb. $\bar{z}$ i.
Thrice daily. Especially
where pains are worse at night.
2. R Guaiacol carbonate gr. v.

In cachets, thrice daily, for
rheumatoid arthritis. Dose to
be gradually increased. Con-
tinue for at least one year, simul-
taneously with:—

- R Potassii iodidi gr. v.-x.
Tr. nucis vom. $\mathbb{M}$ v.
Infusi gent. co. $\bar{z}$ i.
Thrice daily.
 3. R Liq. sodii arseniat. $\mathbb{M}$ iii.
Vini colchici $\mathbb{M}$ x.
Tr. cinchon. co. $\bar{z}$ i.
Aquam ad $\bar{z}$ i.
Thrice daily after meals. In
rheumatic gout.
 4. R Sodii sulphat.
Sulphuris præcip. āā $\bar{z}$ ss.
Every morning in a tumbler-
ful of milk and water.
 5. R Ammon. chlorid. gr. x.-xx.
Infus. gentian. co. $\bar{z}$ i.
Thrice daily. Especially in
muscular rheumatism.
 6. R Quininæ sulph. gr. i.
Strychn. sulph. gr. $\frac{1}{36}$.
Acidi arseniosi gr. $\frac{1}{36}$.
Glycer. tragac. q.s.
M. Ft. pil. i.
Thrice daily. A useful pill in
rheumatoid arthritis.
 7. Ung. ichthyol 50 per cent.
well rubbed into painful
joints, often gives great
relief.
 8. *Turkish baths.*
 9. *Cod-liver oil.*
- Rickets.**
1. *Cod-liver oil* should be given
in small doses ($\mathbb{M}$ xx.- $\bar{z}$ ss.) at
first.
 2. Syr. ferri phosph. co. $\bar{z}$ ss.-i.
or Syr. ferri iodid. $\mathbb{M}$ xx.- $\bar{z}$ ss.
or Vini ferri $\bar{z}$ ss.-i.
In water thrice daily.

3. R Calcii phosph. gr. v.
Bismuth nitrat. gr. v.
Sacch. albi. $\bar{3}$ ss.
Twice daily. Especially with
tendency to diarrhœa.

Scarlet Fever. (*Children.*)

1. *Tr. aconiti*.—One drop every
hour or oftener, at outset of
fever.

2. R Liq. ammon. acet. $\mathbb{M}$ xv.
Tr. aconiti $\mathbb{M}$ ii.
Aquam ad $\mathbb{M}$ ii.

Every two or three hours in
early stages.

- or R Sodii salicylat. gr. ii.
Syrupi aurant. $\mathbb{M}$ xx.
Aquam ad $\bar{3}$ i.

Every hour during day, and
every two hours during night.
Very useful in early stages.

3. General inunction of the body
with equal parts of eucalyptus
oil and olive oil. Repeat
as required. Useful
from outset.

4. *Tepid sponging and baths*, fol-
lowed by inunction. In
early delirium, and during
desquamative stage.

5. For local applications to
throat :—

Sol. hydrarg. biniodid. 1 in
1000.

or Sol. acid. boracic. satur-
ated (sweetened with
glycerine).

or Sol. acid. carbolic. 2 to 3
per cent.

To be used as sprays. If
used for the nares, should be half
of the above strengths.

6. R Tr. ferri perchlor. $\mathbb{M}$ v.
Glycerini $\bar{3}$ ss.
Aquam ad $\bar{3}$ ss.

Every three or four hours. In
scarlatina anginosa. Stimulants
and good food also needed.

7. For scarlatinal nephritis :—
Warm baths.

Saline aperients.

- R Potass. bitart. gr. x.-xx.
Vini ipecac. $\mathbb{M}$ v.
Sp. æther. nit. $\mathbb{M}$ xv.
Infusi digitalis $\bar{3}$ ss.
Aquam ad $\bar{3}$ ss.

(*Children above five years.*)

Three times daily.

Sleeplessness.

1. *Opium*.—Dose to be regulated
according to age. Chiefly
indicated in sleeplessness
from pain. Opium should
not be given for chronic
sleeplessness unaccompa-
nied* by pain, or where
there is albuminuria, nor
in any severe toxic condi-
tion.

2. *Bromide of potassium*.—Dose
gr. xv.-xxx. or more. Use-
ful in sleeplessness from
worry, irritation, and ex-
citement. Often advan-
tageously combined with
chloral.

3. *Chloral hydrate*.—Dose gr.
xv.-xxx. or ii.-v. in chil-
dren. Useful where opium
is contra-indicated. Should
be avoided or given with
caution where the circula-
tion or respiration is em-
barrassed.

4. *Alcohol* with light nourishment
of great service in the sleep-
lessness of the aged and
feeble. Also in certain low
and malignant types of
fever. To be taken at bed-
time.

5. *Hyoscyamus*.—The tincture
($\mathbb{M}$ v.-x.) is a useful sedative
for young children. May
be combined with pot.
brom. and camphor water.

6. *Hyscine hydrobromate*.—Subcutaneous injection of gr. $\frac{1}{200}$ or more. Useful in mania, and wild delirium.

7. *Sulphonal* gr. x.-xxx.

or *Trional* gr. x.-xx. Dissolved in a little whisky or brandy and boiling water. To be taken with some warm food an hour before bed-time.

8. *Paraldehyde*—in doses of $\bar{3}$ i. or more, dissolved in water. Not being very soluble a better form is:—

R Paraldehyde $\bar{3}$ i.
Mucilag. acaciæ $\bar{3}$ ii.
Syrupi simp. $\bar{3}$ ii.
Aquam cinnamomi ad $\bar{3}$ iss.

A very safe hypnotic. Especially useful in cardiac cases.

9. A tumblerful of hot water, or peppermint water, with $\bar{3}$ i. of soda dissolved in it. At bed-time. Often useful in insomnia from flatulent dyspepsia.

Small-pox.

1. R Sodii salicylat. gr. xv.
Ammon. carb. gr. iv.
Aquæ $\bar{3}$ i.

Every three or four hours. In early stages (adults). Later on add ferri et am. cit. gr. v. to each dose. A tepid bath each day; after which pocks to be painted over with:—

R Acid. carbolic. $\bar{3}$ i.
Glycerini $\bar{3}$ i.
Gelatini $\bar{3}$ i.
Aquæ $\bar{3}$ xxvi.

During exfoliation a warm, slightly alkaline bath daily. (*Prideaux*.)

2. Phenazon. gr. v.

Every three or four hours. To relieve pains in the back.

3. R Quin. sulph. gr. ii.
Tr. ferri perchlor. $\mathbb{M}$ xii.
Aq. chloroformi $\bar{3}$ i.

Every four hours where there is much debility and prostration. Also good food and alcohol.

4. R Calaminæ $\bar{3}$ i.
Olei eucalypti $\bar{3}$ ss.
Lin. calcis ad $\bar{3}$ viii.

To be brushed over the face to prevent pitting, and to relieve irritation.

Stomach, Ulcer of.

1. R Bismuthi carb.
Sodii bicarb. āā gr. v.
Acid. hydrocyan. dil.
 $\mathbb{M}$ iii.
Aq. et mucilag. ad $\bar{3}$ ss.

Three or four times daily. To relieve pain and vomiting. Liq. morph. may be often added with benefit.

2. R Liq. arsenicalis $\mathbb{M}$ i.-ii.
Recommended by Ringer to check pain and vomiting. To be taken in water before food.

3. R Argenti nitrat. gr. $\frac{1}{4}$.
Ext. opii gr. $\frac{1}{2}$.
Ext. hyoscyam. gr. ii.
Ft. pil.

Thrice daily.

4. *Sulphate of soda*—in aperient doses, or in form of Carlsbad salt, to be taken early in morning to clear out stomach.

5. *Nutrient enemata*, e.g.
Milk, 4 ounces.
Yolk of 1 egg.
Salt.

The addition of one teaspoonful of liq. pancreaticus (Benger) is desirable; also if the rectum be irritable, $\mathbb{M}$ v.-x. of laudanum.

Stomatitis.

1. R Potass. chlorat. gr. v.
Tr. ferri perch. ℥v.
Glycerini ℥i.
Aquam ad ℥ss.
Three, four, or more times a day (℥i.-ii. for children) in all forms of stomatitis.
2. *Glycerine of borax* or *boroglyceride*—good local applications in many forms.
3. R Liq. hydrogen. peroxid. ℥i.
Aquam ad ℥viii.
A useful mouth wash.
4. *Glycerine of tannin*—as a local application in ulcerative stomatitis.

Syphilis.

1. *Mercury*—in primary and secondary syphilis. The following are some of the chief forms of administration :—

R Hydrarg. cum cretâ gr. ¼-i.
As a powder with sugar, or a pill with ext. gentian. three times a day.

R Pil. hydrarg. subchlor. co.
gr. v.

Twice or thrice daily.

R Ung. hydrargyri ℥i.

To be used as an inunction daily.

R Hydrarg. subchlor. gr.
xv.-xxx.

Volatilised by heat, and used with vapour bath.

N.B.—As soon as the gums are in the least affected, smaller doses should be taken. Slight mercurial treatment advisable for at least two years after primary sore, in order to eradicate disease.

2. R Liq. hydrarg. perch. ℥i.
Decocti sarsæ co. vel
Decocti cinchon. ℥i.
Thrice daily in chronic cases.

3. R Potass. iodid. gr. v.
Ammon. carb. gr. ii.
Liq. hydrarg. perch. ℥i.
Inf. gentian. ℥i.
Thrice daily. A useful formula.

or R Potass. iodidi gr. v.-xv.
Sp. ammon. arom. ℥xx.
Inf. gentian. co. ad ℥i.
Thrice daily, in tertiary lesions.

4. R Hydrarg. perchlor. gr. i.
Glycerini boracis ℥i.
To be painted on obstinate secondary ulcers of mouth. Added to half a pint of water, may be used as a gargle.

5. R Liq. Donovan. ℥x.-xv.
Aquam ad ℥i.
Thrice daily. Especially in scaly skin affections.

6. General tonics and *cod-liver oil*, preferable at first to specific treatment in long-standing cachectic cases.

Typhoid Fever. (See Fever.)

1. *Bath* of 95° cooled to 85° and continued for twenty minutes. To be repeated as often as necessary. For high fever. Most useful in early stages.

2. *Sponging the body* with tepid water, ice being applied to head.

In extreme restlessness, delirium, and wakefulness.

3. Cold applications to head.

R Phenacetin. gr. v.-x.

vel R Acetanilid. gr. ii.-v.

For severe headache. Repeat as required.

4. R Potassii chlorat. gr. xxx.
Acidi hydrochlor. fort.
℥xl.

Mix in a ℥xii. bottle, add water gradually, and shake after each addition till the bottle is full.

Then add quinine (12 to 24 grains). Two tablespoonfuls for a dose.

5. R Hydrarg. subchlor. gr. $\frac{1}{4}$.
 R β -naphthol gr. v.-x. In cachets.
 R Salol. gr. v.-x. In cachets.
 R Acid. carbolic. $\mathbb{M}$ i.-ii. (diluted).
 R Bismuthi salicylat. gr. x.-xv.

Useful intestinal antiseptics. To be given every four or six hours.

6. Enema of hot borax solution (1 per cent.) followed by enema of tr. opii $\mathbb{M}$ xx. in gruel.

or Pulv. ipecac. co. gr. ii.—every four hours.

or Pil. plumbi c. opio gr. ii.—every four hours.

or Chlorine mixture without quinine (No. 4).

For diarrhœa. Attend to diet.

7. Mist. ol. ricini (B.P.) $\mathfrak{Z}$ ss.-i.
 At intervals, as required.

or Enemata.

For constipation.

8. R Ol. terebinth. $\mathbb{M}$ x.
 Mist. amygdalæ $\mathfrak{Z}$ i.

Every three or four hours. An excellent formula for routine use in all cases; but especially towards end of second week, with dry, brown, cracked tongue, and tympanitis. Also in hæmorrhage.

9. R Ol. terebinth. $\mathfrak{Z}$ i.
 Ol. olivæ $\mathfrak{Z}$ iii.
 Sp. ammon. foëtis. $\mathfrak{Z}$ ss.
 Decoct. amyli ad Oi.

A useful enema for severe tympanitis.

10. *Strong black coffee*—two or three tablespoonfuls occasionally.

R Strychninæ nitrat. gr. $\frac{1}{30}$.
 Subcutaneously—as required.

R Sp. ammon. arom. $\mathbb{M}$ xx.
 Sp. ætheris $\mathfrak{Z}$ ss.
 Tr. lavand. co. $\mathfrak{Z}$ ss.
 Aq. camph. ad $\mathfrak{Z}$ i.

Three or four times a day.
 Useful cardiac stimulants.

11. R Sulphonal *vel* trional gr. x.-xx.

Dissolved in whisky and hot water. For insomnia.

12. R Liq. opii sed. $\mathbb{M}$ x.-xx.
 Sp. æth. nit. $\mathfrak{Z}$ ss.
 Sp. ammon. arom. $\mathfrak{Z}$ ss.
 Aq. camph. ad $\mathfrak{Z}$ i.

For delirium and general restlessness—if opium is not contra-indicated.

13. R Acid. tannic. $\mathfrak{Z}$ i.
 Sp. vini meth. $\mathfrak{Z}$ vi.

Lotion for preventing bed-sore.

14. R Acid. boric. $\mathfrak{Z}$ i.
 Glycerini $\mathfrak{Z}$ i.
 Lemon juice q.s.
 Aquam ad $\mathfrak{Z}$ iv.

Uræmia.

1. *Packing*—with blanket wrung out in hot water to which a little mustard has been added.

2. Vapour-baths, and hot diluent drinks.

3. *Pilocarpine*—gr. $\frac{1}{8}$ - $\frac{1}{4}$ injected subcutaneously. Not suitable for children; nor in cases of extreme anasarca.

4. Pulv. jalapæ co. $\mathfrak{Z}$ i.

or Saline aperients.

or Pulv. elaterini co. gr. ii.

Useful purgatives in uræmia. Repeat as required.

5. Sour butter-milk in small quantities at a time. For uræmic vomiting (Whitla).

6. R Ammonii bromid.
Chloral hyd. āā ʒss.
Warm water ʒii.

Useful rectal injection in uræmic convulsion.

- or Inhalations of pure oxygen.
7. Erythrol tetranitrat. gr. $\frac{1}{2}$ (in tablet).
or Trinitrin gr. $\frac{1}{100}$ (in tablet).
or Nitrite of amyl—for inhalation.

For uræmic dyspnœa.

8. Subcutaneous injection of half a pint (or more) of normal saline solution. For uræmic coma.

Urine, Incontinence.

1. Attend to thread-worms, vesical calculus, adherent or long prepuce—if present. Especially in children.
2. *Belladonna*—Ten to fifteen drops of the tincture thrice daily, with camphor water. In simple incontinence of children.
3. Ext. rhus. aromat. liq. ℥v. if under two years old, otherwise ℥x., night and morning.
4. R Liq. arsenicalis ℥i.
Tr. nucis vom. ℥i.
Glycerin ℥xx.
Aquam ad ʒi.

Thrice daily, after meals for a child of 5–10 years.

5. Tr. cantharidis ℥i., three or four times a day, useful for incontinence in adult females occurring on exertion or sudden movement of any kind.
6. R Sodii salicylatis gr. v.
Sodii citrat. gr. x.
Tr. belladonnæ ℥v.
Syrupi aurantii ʒi.
Aq. ad ʒss.

Thrice daily. For incontinence associated with highly acid urine.

7. *Tonics*.—Especially iron and strychnia.

Vomiting.

1. R Sodii bicarb. gr. iii.
Tr. nucis vom. ℥ $\frac{1}{2}$.
Sp. chloroform. ℥i.
Aq. et syrup. ad ʒi.

Three or four times daily. Useful in chronic vomiting in children with gastric catarrh.

2. R Tr. nucis vom. ℥i.
Every half or one hour as required.

In chronic vomiting of nervous women with indigestion.

3. R Bismuth. carb. gr. v.-x.
Sodii bicarb. gr. x.
Acid. hydrocyan. dil. ℥iii.
Mist. amygdalæ ʒi.

Thrice daily in some forms of gastric vomiting. Liq. morph. may be added if much pain, as in cancer of stomach; or sp. ammon. aromat. if too "cold."

4. R Tr. iodi ℥i.
Glycerin. acid. carbolic. ℥v.
Aquam ad ʒi.

Every three or four hours in fermentative vomiting.

5. R Liq. arsenicalis ℥i.

In water before food, in vomiting of drunkards (Ringer).

6. R Vin. ipecac. ℥i.

Every half or one hour in water as required. In almost all cases of reflex vomiting.

Whooping Cough.

1. *Salines* and *expectorants* in catarrhal stage.
2. R Acid. carbolic. ℥i.
Ess. menth. pip. ℥ $\frac{1}{2}$.
Syrupi ʒss.
Mucilaginis ʒss.

Every four or six hours. A very useful formula; may be combined as required with tr. belladon. ℥v., or tr. camph. co. ℥v.

For children from five years and upwards.

3. R Potass. bromid. gr. i.-v.
Tr. belladon. ℥v.-x.
Syrupi tolut. ℥xx.
Aquam ad ʒi.

or R Phenazon. gr. i.-iii.
Ammon. bromid. gr. ii.
Syrupi tolut. ℥xx.
Aq. ad ʒi.

Every four or six hours. For a child of four years and upwards. Children bear belladonna very well.

4. R Aluminis gr. ii.
Syrupi limonis ℥xx.
Aq. ad ʒi.

Every three or four hours. In cases with profuse secretion.

5. R Resorcin gr. ii.
Tr. belladon. ℥v.
Syrupi tolut. ℥xx.
Aq. ad ʒi.

Every three or four hours.

6. *Counter-irritation* to chest. A valuable adjuvant, if much bronchial secretion. Turpentine liniment is a convenient preparation.

7. Vaporisation of 5 per cent. solution of crude carbolic acid, or of creolin, over a lamp, near bedside.

Worms.

1. R Ext. filicis liq. ʒi.

Ovi vitelli.

Syrupi ʒss.

Aq. chloroformi ad ʒii.

For tape-worm. To be given as directed in text. Half-dose for children.

or The extract may be given in capsules (gr. xv.) every ten minutes for four doses. In either case, follow up with full dose of calomel three or four hours later.

2. R Pelletierin tannate gr.v.-x.

Suspended in water, or given in capsule. Useful for tape-worm. Not suitable for children.

3. R Santonin gr. ii. in castor oil.

For round worms. Double the dose for adults. Or may be given in syrup of senna. Or in the form of lozenges B.P.

4. Enemata of salt solution ʒi. to Oss. or of Inf. quassia.

For thread-worms. A little mercurial ointment just inside the anus will prevent migration of worms at night.

DISEASES OF THE SKIN.

Aene.

1. R Sulphur. præcip. ʒi.
Calamina ʒii.
Zinci oxidi ʒiii.
Glycerini ʒii.
Aquam ad ʒvi. Ft. lotio.
Useful with much irritation.

2. R Sulphur. præcip. ʒss.
Acid. salicylic. gr. x.
Vaselini ʒi. Ft. ung.

For head and face.

3. R Resorcini ʒii.
Zinci oxidi.
Amyli aa ʒii.

Apply three successive nights in the week, in obstinate cases.

4. Vaccines are useful in chronic cases.

Alopecia areata.

1. R Aceti cantharidis ℥i.
Ung. hydrarg. ox. rub. ℥i.

Ft. ung. To be used after washing with soap and hot water.

2. Ung. sulphuris.

3. R Ol. cadini ℥ss.
Sulphuris ℥ss.
Vaseline ℥i. Ft. ung.

Boils.

1. Lotio acid. carbolic. 1 in 40.
or Sol. hydrarg. perchlor. 1 in 1500.

To cleanse the adjacent skin to prevent reinfection.

2. Unna's carbolic mercury plaster.

3. Fresh brewer's yeast. Half a wineglassful twice daily.

4. Calcii sulphide gr. $\frac{1}{4}$ to $\frac{1}{2}$.
In coated pill. Three times daily.

5. Staphylococcic vaccines are useful in chronic cases of boils and coccogenic sycosis.

Chilblains.

1. Tinct. iodi.
Ol. terebinth. æq. partes.

or R Ichthyol.
Acid. tannic.
Resorcin. āā ℥i.
Aquæ ℥v. Ft. lotio.

To be painted on at night.

2. Parts to be rubbed with equal parts of tr. capsici and eau de Cologne; or lin. terebinth. acet.

3. R Ung. acid. borici.
or R Balsam. Peruv. ℥i.

Ung. zinci ad ℥i. Ft. ung.
Good applications for broken chilblains.

Corns.

R Acid. salicylic ℥i.
Ext. cannabis ind. gr. v.
Collodion flexilis ℥i.

To be painted on with a brush daily.

Eczema.

ACUTE.

1. Vini antimonialis ℥x.
Three or four times daily.
Often useful with salines.

2. R Calaminæ ℥iii.
Zinci oxidi ℥ii.
Glycerini ℥ii.
Aq. calcis ad ℥vi. Ft. lot.

or R Liq. plumbi subacet. ℥ss.
Lactis Oss. Ft. lotio.

Useful sedative lotions when discharge is marked.

3. R Acid. salicylic. gr. x.
Zinci oxidi.
Amyli āā ℥ii.
Vaseline ℥ss. Ft. pasta.

Lassar's paste. May be used for dry or moist cases when discharge is moderate.

CHRONIC.

1. Removal of scales, crusts, or scabs by soaking with olive oil or weak alkaline solutions.

2. If surface discharges, lotions are better than ointments.

3. Ung. hydrarg. ammon. (gr. v. or x. to ℥i.). Useful for scaly patches with moderate hyperæmia.

4. R Acidi salicylic. gr. x.
Olei cadini ℥ss.
Ung. zinci ox. ℥i. Ft. ung.

Useful in irritable papular cases, with considerable thickening.

5. R Sulphur. præcip. gr. x.-xx.

Ung. zinci ox. $\frac{3}{4}$ i. Ft. ung.

For seborrhœic eczema.

6. R Argenti nitrat. gr. x.

Sp. ætheris nit. $\frac{3}{4}$ i. Ft. pig.

For eczema rimosum and perineal eczema.

Erythema.

1. R Camphoræ gr. xx.

Sp. vini rect. q.s.

Acidi borici $\frac{3}{4}$ ii.

Zinci oxidi.

Amyli $\frac{1}{2}$ ss.

Useful dusting powder.

2. R Cere.

Cetacei $\frac{1}{2}$ ss.

Ol. amygdalæ.

Aq. rosæ $\frac{1}{2}$ ss.

Ft. ung. "cold cream."

or R Lanolin. $\frac{3}{4}$ i.

Vaselini $\frac{3}{4}$ ii.

Liq. plumbi subacet. $\frac{3}{4}$ iv.

Ft. ung.

Favus.

1. Remove crusts by boracic poultice, or carbolised oil.

2. Epilate by forceps or X-rays.

3. Wash well with soft soap.

4. R Cupri sulphat. $\frac{3}{4}$ i.

Ung. zinci ox. $\frac{3}{4}$ i. Ft. ung.

5. Paint frequently with Tr. iodi.

Herpes zoster.

1. Parts may be protected by painting with collodion.

2. Calamine lotion. Useful sedative.

3. R Potass. bromidi.

Phenazoni $\frac{1}{2}$ ss. gr. x.

Liq. morph. hyd. $\frac{1}{2}$ x.

Aquam ad $\frac{3}{4}$ i. Ft. haust.

For sleeplessness caused by pain.

Impetigo contagiosa.

Remove scabs by poultice or oil, and apply ung. hydrarg. ammon. (gr. x. to $\frac{3}{4}$ i.).

Lichen planus.

1. Liq. arsenicalis $\frac{1}{2}$ iii. (gradually increased) thrice daily, in water.

2. R Potass. iodid. gr. v.

Liq. hydrarg. perch. $\frac{3}{4}$ ss.

Decoct. sarsæ co. $\frac{3}{4}$ i.

Thrice daily.

3. R Resorcin. gr. xx.

Ung. hydrarg. am. (half

B.P. strength) $\frac{3}{4}$. Ft.

ung.

For local application.

Lupus.

1. Lotio nigra. Useful dressing for ulcerated surfaces.

2. Paint discharging and ulcerated areas with pure carbolic acid.

3. R Acid. salicylic. gr. xx.-xxx.

Creosoti $\frac{1}{2}$ x.

Collodion flexilis $\frac{3}{4}$ i.

Ft. pigment. Ulcers thus formed to be healed under ung. acid. borice.

4. Acidi pyrogallici gr. xxx.-xl.

Vaseline $\frac{3}{4}$ i.

Useful for dry nodular cases.

Lupus erythematosus.

1. Calamine lotion if much irritation.

2. Lassar's paste (*v. Eczema*).

Pediculosis.

1. Ung. hydrarg. ammon. Useful in all forms. Dilute if associated with much eczema or pustulation.
2. Lotio acid. carbolie. (1 in 40).
3. To remove nits from hair, wash with 25% sol. of acetic acid, and follow up with fine tooth comb.
4. Ung. staphisagriæ. Best application for P. corporis. Clothes to be disinfected.

Pemphigus.

1. Liq. arsenicalis, increasing doses.
2. Ruptured bullæ to be treated with boric acid ointment or dusting powder.

Pruritus.

1. Warm alkaline baths, or mild alkaline lotions.
2. R Acid. carbolie. $\mathfrak{z}$ i.
Liq. potassæ $\mathfrak{z}$ ss.
Aquam ad Oss. Ft. lotio.
3. Liq. carbonis deterg. $\mathfrak{z}$ ii.-iv.
Aquam ad Oss. Ft. lotio.
4. Lib. plumbi subacet. $\mathfrak{z}$ ii.
Glycerini $\mathfrak{z}$ ss.
Aquam ad Oss. Ft. lotio.
5. Ung. conii. Invaluable for pruritus ani; with occasional local application of sol. argent. nit. (gr. 20 to $\mathfrak{z}$ i.).
6. Thorough disinfection with mercuric perch. sol. (1 in 2000), followed by application ung. acidi carbolie. (3%). For pruritus vulvæ.

Psoriasis.

1. Vin. antimonialis $\mathfrak{M}$ x.
Thrice daily, in very acute cases.

2. Ung. chrysarobin. P.B.

To be firmly rubbed into the patches. Unsuitable for the face.

3. R Chrysarobin. gr. x.-xxx.
Liq. gutta-perchæ $\mathfrak{z}$ i.

To be painted over patches. Not to be used for the face.

4. Ung. picis B.P.

5. R Liq. carbonis deterg. $\mathfrak{M}$ x.
Hydrarg. ammon. gr. x.
Adipis benzoat. $\mathfrak{z}$ i. Ft. ung.

6. R Ol. cadini $\mathfrak{M}$ xx.

Ung. zinci $\mathfrak{z}$ i. Ft. ung.
Useful if much inflammation, or if the skin is cracked.

Ringworm.

1. Rub in Tr. iodi., afterwards using ung. sulphuris.
2. R Sulphur. præcip.
Hydrarg. ammon. $\mathfrak{a}\mathfrak{a}$ $\mathfrak{z}$ ss.
Acid. salicylic. gr. xv.
Ol. olivæ $\mathfrak{z}$ iii.
Lanolin. ad $\mathfrak{z}$ i. Ft. ung.
3. R Iodini $\mathfrak{z}$ ii.
Colourless oil of tar $\mathfrak{z}$ vi.
(Coster's paint.) Apply with a stiff brush.
4. Ung. hydrarg. oleat. 5 to 10%.
5. Epilate with the X-rays in scalp or beard cases, unless there is much inflammation.

Rosacea.

1. R Sulphur. præcip. $\mathfrak{z}$ ss.-ii.
Calaminæ $\mathfrak{z}$ iv.
Glycerini $\mathfrak{z}$ ii.
Aq. calcis ad $\mathfrak{z}$ vi. Ft. lot.
2. R Ichthyol $\mathfrak{z}$ i.
Olei olivæ $\mathfrak{z}$ iii.
Lanolin. ad $\mathfrak{z}$ i. Ft. ung.

Scabies.

Thoroughly wash and rub body with soft soap, and hot

water. Dry with rough towel, and afterwards use

1. Ung. sulphuris ($\frac{1}{2}$ B.P. strength).
2. Boil 2 oz. of sublimed sulphur with 1 oz. of slaked lime in a gallon of water. Apply with a small sponge. Never fails, but if used too frequently will set up irritation.
3. *Tonics* and *cod-liver oil* needed in pustular forms, with a mild ointment such as Balsam of Peru $\mathfrak{z}$ i.-ii. to $\mathfrak{z}$ i. of ung. zinci.

Seborrhœa capitis.

1. Wash the scalp well with sulphur or carbolic soap, or plain borax and hot water.
2. R Sulphuris præcip. gr. xx.
Acid. salicylic. gr. x.
Vasellini $\mathfrak{z}$ i. Ft. ung.
3. R Liq. carbonis deterg. $\mathfrak{z}$ i.
Hydrarg. ammon. $\mathfrak{z}$ ss.
Vasellini $\mathfrak{z}$ iss. Ft. ung.

If associated with eczema of scalp.

Tinea versicolor.

1. Sodii hyposulphitis $\mathfrak{z}$ ss.
Aq. Oss. Ft. lotio.
- or Ung. sulphuris ($\frac{1}{2}$ B.P. strength).
Use after thorough washing.

Urticaria.

1. *Calomel*, or Hyd. $\bar{c}$ cretâ at night, followed by saline aperient next morning.
2. *Tonics*, especially *quinine*, in chronic cases.
3. Salicylate of soda, with bicarbonate, in rheumatic cases.
4. Calcium lactate in 15 grain doses three times a day for two days followed by periods of intermission of two or three days.
5. Anti-pruritic lotions.

APPENDIX B.

SCHEME FOR ANALYSIS OF GASTRIC CONTENTS.

1. Take 10 c.c. of filtered contents in small glass beaker. Add decinormal solution of soda from a burette till a drop of the "contents" (taken out with a platinum loop) no longer gives a red colour when added to a half per cent. solution of dimethyl-amido-azo-benzol in alcohol. Multiply amount of solution of soda employed (in c.c.) by '0365. The result gives the percentage of free HCl.

2. Apply a droplet of the contents to a slip of Congo-red test paper. If a greyish-blue colour is produced, free organic acids are present. Add decinormal solution of soda till this reaction no longer occurs. Note the amount of soda solution employed. Multiply by '0365. The result gives the percentage of free organic acids in terms of HCl.

3. Add decinormal solution of soda now to the contents until a droplet gives a violet colour with a one per cent. solution of alizarin. (The tint should be compared with that formed by the addition of three or four drops of the alizarin solution to 5 c.c. of one per cent. solution of sodium carbonate.) Multiply the amount of sodium solution used by '0365. The result gives the percentage of acid salts present.

4. To the remainder of the contents add a drop or two of a one per cent. solution of phenolphthaline, and afterwards add decinormal solution of soda till a permanent rose-pink is obtained. The amount of soda solution employed multiplied by '0365 gives the percentage of combined HCl.

5. The total quantity of soda solution employed from first to last multiplied by '0365 gives the percentage of total acidity.

WIDAL'S REACTION.

The principles which underlie this reaction have been referred to in the text (p. 40). When the reaction is carried out with a culture of living typhoid bacilli it is essentially a laboratory procedure associated with microscopic examination; but for ordinary clinical purposes a permanent sterile suspension of dead bacilli will serve, and the results can be observed with the naked eye. No complicated apparatus is required, and the technique is sufficiently simple to be carried out by the practitioner himself. Parke, Davis & Co. supply a convenient and inexpensive outfit for the purpose, which they call a Typhoid Agglutometer. A little blood is withdrawn into a tube, and the serum is allowed to separate. Two drops of this serum are added to a given quantity of diluting fluid by means of a pipette, yielding a 1 in 5 dilution of serum. A given quantity of the serum-dilution is added to each of three tubes representing different strengths of suspended bacilli. After a time, when the reaction is positive, floccules appear in one or more of the tubes, depending upon the agglutinating power of the serum which is being tested, and detection of the change is facilitated by comparison with another tube for control. Full directions for use accompany each outfit.

DIAZO-REACTION.

Ehrlich's diazo-reaction is ordinarily present in well-developed attacks of typhoid fever, but may be absent in mild cases. It

generally appears when the temperature has reached its height at the end of the first week, and persists for about a fortnight. It is not confined to typhoid fever, and is often found in rapidly progressive acute miliary tuberculosis, and occasionally in other specific febrile disorders. The following reagents should be freshly prepared: (a) A saturated solution of sulphanilic acid to which 5 per cent. of hydrochloric acid should be added; (b) a half per cent. solution of sodium nitrite. To a small quantity of urine in a test-tube add an equal volume of the sulphanilic solution, and then three or four drops of the nitrite solution. Alkaline with ammonia and shake vigorously. A pink froth is formed when the reaction is positive; and, on standing, the fluid deposits a dark green precipitate. The reaction is of small diagnostic significance.

DETECTION OF TUBERCLE BACILLI IN SPUTA.

Select a small purulent fragment from the sputa, and place between two glass cover-slips. By sliding one over the other with a little pressure, a thin film is spread out over each. Allow the slips to dry, and then pass them quickly through the flame of a spirit-lamp. Then holding one with forceps (face downwards) let it drop upon the surface of the following staining solution in a porcelain capsule: Carbolic acid 5 grms., alcohol 10 c.c., fuchsin 1 gm., distilled water 100 c.c. Warm over a spirit-lamp till steam begins to rise, and continue for four or five minutes. Wash the slip in water, decolorise in 25 per cent. solution of hydrochloric acid, and again wash. Afterwards stain with methylene blue for ten or fifteen seconds; wash out superfluous stain and dry, and mount in a drop of Farrant's solution or Canada balsam, and examine in a good light with a high power, preferably using $\frac{1}{2}$ inch immersion-lens, and a sub-stage condenser. Tubercle bacilli, if present, are stained a bright red colour.

TEST FOR OCCULT BLOOD IN FÆCES.

Add a small portion of the suspected faecal matter to a few centimetres of water in a test-tube, and boil. In another test-tube add ten to fifteen drops of a saturated solution of benzidin in glacial acetic acid to two or three c.c. of a ten-volume solution of peroxide of hydrogen. On addition to the latter of a few drops of the faecal solution, a blue or bluish-green colour will develop, if blood be present.

A still simpler but not so delicate a way of applying the same test is to use filter paper which has been soaked in a saturated solution of benzidin in glacial acetic acid, and afterwards dried. If a piece of such paper be dipped into the faecal solution, and then treated with a ten-volume solution of hydrogen peroxide, the paper will show a blue coloration in the course of a minute or two, if blood be present.

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